A STUDY OF COMPARISON OF LEFT VENTRICULAR MASS IN HYPERTENSIVE PATIENTS WITH DIABETES MELLITUS AND WITHOUT DIABETES

A Dissertation Submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI

In Partial Fulfilments of the Regulations

for the Award of the Degree of

M.D. (GENERAL MEDICINE) - BRANCH – I

GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI

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BONAFIDE CERTIFICATE

This is to certify that the Thesis- **"A study of comparison of left ventricular mass in hypertensive patients with diabetes mellitus and without diabetes mellitus"** is a genuine work done by Dr.S.Krishnamoorthi, Post-graduate student in Department of General Medicine, Government Kilpauk medical college, Kilpauk, Chennai, under the guidance of Prof. Dr. N. GUNASEKARAN, M.D., DTCD, Head of the Department of General Medicine, Govt. Kilpauk Medical College.

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DECLARATION

I, **Dr.S.KRISHNAMOORTHI**, solemnly declare that the dissertation titled **"A study of comparison of left ventricular mass in hypertensive patients with diabetes mellitus and without diabetes mellitus"** has been prepared by me. This is being submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of MD degree Branch I (General Medicine).

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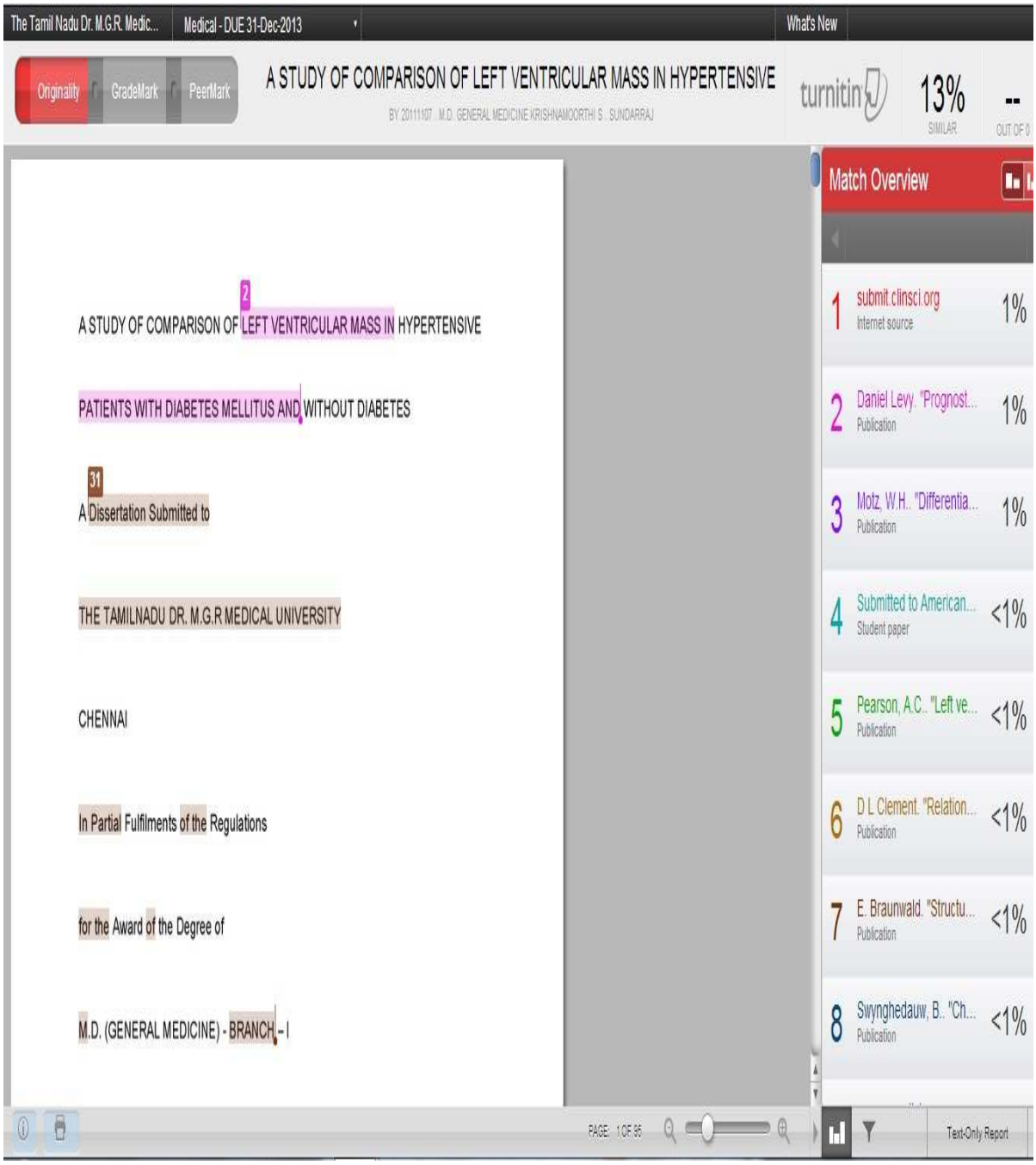
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ABSTARCT

BACKROUND AND OBJECTIVES:

Hypertension is a major risk factor for stroke, cardiovascular diseases and aortic dissection and hypertension is associated with significant morbidity and mortality. Hypertension causes 7.1 million premature deaths and the disease burden is 4.5% all over the world. Hypertension is a major public health problem globally. The prevalence of diabetes has raised to 285 million in 2012 when compared to 30 million cases in 1985. The principal morbidity and driver of mortality in patients with diabetes are cardiovascular diseases (CVD). Both hypertension and diabetes increases cardiovascular morbidity and mortality by increasing left ventricular mass. . Even though the ECG can identify the findings suggestive of left ventricular hypertrophy, the sensitivity is very less. So echocardiography is preferred for assessment of left ventricular hypertrophy.

MATERIALS AND METHODS:

This was a prospective cross sectional study conducted in patients attending the hypertension OP of Govt. Kilpauk Medical College Hospital with hypertension and diabetes or hypertension alone of more than 5 years duration without any cardiac disease and healthy individuals of more than 35yrs of age

are taken as controls. Echocardiography was done for both study and control groups and left ventricular mass was calculated by using Penn formula and LV mass was compared between the three groups (HT, HT and DM, controls).

RESULTS:

There is significant statistical correlation between age group and left ventricular mass in hypertension with diabetes group (p =0.039). There is significant statistical correlation between females (p=0.042) with diabetes and hypertension and severity of abnormal left ventricular mass than males (p=0.286). The mean left ventricular mass increases in both males (211.25gm-HT to 224.24gm-HT+DM) and females (169.65gm-HT to 206.06gm-HT+DM) in hypertension and diabetes group and it was statistically significant in females (p=0.015) but not in males (p=0.546).

CONCLUSION:

The study shows that there is increased left ventricular mass in females with hypertension and diabetes mellitus when compared to females with hypertension alone but not in males. But the mean left ventricular mass was increased in both males and females of diabetes and hypertension group when compared to hypertension only group. Advancing age is also associated with increased left ventricular mass in cases of both hypertension and diabetes group. Increased left ventricular mass is an important cause for increased

cardiovascular morbidity and mortality hence, the females with diabetes and hypertension should be managed aggressively for reduction of left ventricular mass.

KEY WORDS:

Left ventricular mass, Hypertension, Diabetes mellitus, Echocardiography, Penn formula, Cardiovascular diseases.

I. INTRODUCTION

Hypertension is a major risk factor for stroke, cardiovascular diseases and aortic dissection and hypertension is associated with significant morbidity and mortality. The WHO estimates that hypertension may cause 7.1 million premature deaths and the disease burden 0f 4.5% all over the world. Hypertension is a major public health problem globally.

The development of echocardiography has offered new approaches regarding the pathophysiology and clinical implications that affect the hypertensive patients. Echocardiography is very important in the clinical management of any hypertensive patient. Even though the ECG can identify the findings suggestive of left ventricular hypertrophy, the sensitivity is very less. The sensitivity of the criteria using ECG for LVH was 7% to 35% in moderate hypertrophy and 10% to 50% in severe hypertrophy. So echocardiography is preferred for assessment of left ventricular hypertrophy. In echocardiography, M-mode technique is the gold standard test.

Diabetes mellitus refers to “A group of common metabolic disorders that share the phenotype of hyperglycemia. The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on individuals with diabetes as well as on the health care system”.

Diabetes mellitus also predisposes to cardiovascular diseases. In diabetes mellitus hyperinsulinemia (insulin resistance), high HbA1c and dysautonomia contributes to increased left ventricular mass. Compared to non-diabetic individuals, diabetic individuals have raised morbidity and mortality from cardiovascular disease.

II. REVIEW OF LITERATURE

II. 1. HYPERTENSION:

Hypertension is a major public health problem all over the world. Approximately 7.6 million deaths (13-15% of the total) and 92 million disability adjusted life years worldwide were attributable to high blood pressure in 2001. Hypertension doubles the risk of cardiovascular diseases, including coronary heart disease, congestive heart failure, ischemic and hemorrhagic stroke, renal failure and peripheral arterial diseases. Because of increasing obesity and population aging, hypertension burden is increasing globally and projected to affect 1.5 billion persons (one third of world's population) by the year 2025. By current data, hypertension causes 54% of stroke and 47% of ischemic heart disease throughout the world [(74)]. Antihypertensive therapy reduces the risk of cardiovascular renal diseases.

II. 1. a. Epidemiology:

Prevalence of hypertension, blood pressure levels and the rate of age-related increases in blood pressure vary among countries and subpopulations within a country. Hypertension is less prevalent in individuals living in primitive and culturally isolated societies. In early adulthood the average systolic blood pressure was higher for men in the United States, whereas in ages 60 and above it is higher for women when compared to men.

There is a progressive increase in diastolic blood pressure upto 55 years after which it progressively decreases. As a result, beyond the age of 60, there is widening of pulse pressure. The probability of developing systemic hypertension is 90% for a middle aged or elderly person in his or her lifetime.

II. 1. b. Definition:

Hypertension is defined as "Blood pressure of ≥ 140/90 mmhg" for which there are definitely established benefits from drug treatment which decreases morbidity and mortality [75]. The criterion for defining hypertension was recommended by JNC 7. It has been classified as patients having normal blood pressure, prehypertension and stage 1, stage 2 hypertension.

15 to 20% of patients diagnosed as stage I hypertension based on office blood pressure have ambulatory reading less than 135/85mmhg. This is called as white coat hypertension. It may also be associated with risk of target organ damage and risk of developing sustained hypertension.

Target organ damage is better predicted by home blood pressure including 24-hour blood pressure recording than do office blood pressures. After waking up, the early morning period blood pressure tends to be higher than that other times of day, which explains why myocardial infarction has more common in early morning hours.

BP STAGE	SYSTOLIC BP (MM HG)	DIASTOLIC BP (MM HG)
Normal	<120	<80
Prehypertension	120-139	80-89
Stage 1 hypertension	140-159	90-99
Stage 2 hypertension	≥160	≥100

TABLE 1: Joint National Committee 7(Jnc7) Classification of Blood Pressure (BP) [102]

There is 10 to 20% decrease in blood pressure during night-time when compared to day and when this doesn't happen there is increased risk of cardiovascular diseases.

II. 2. DIABETES MELLITUS

II. 2. a. Definition:

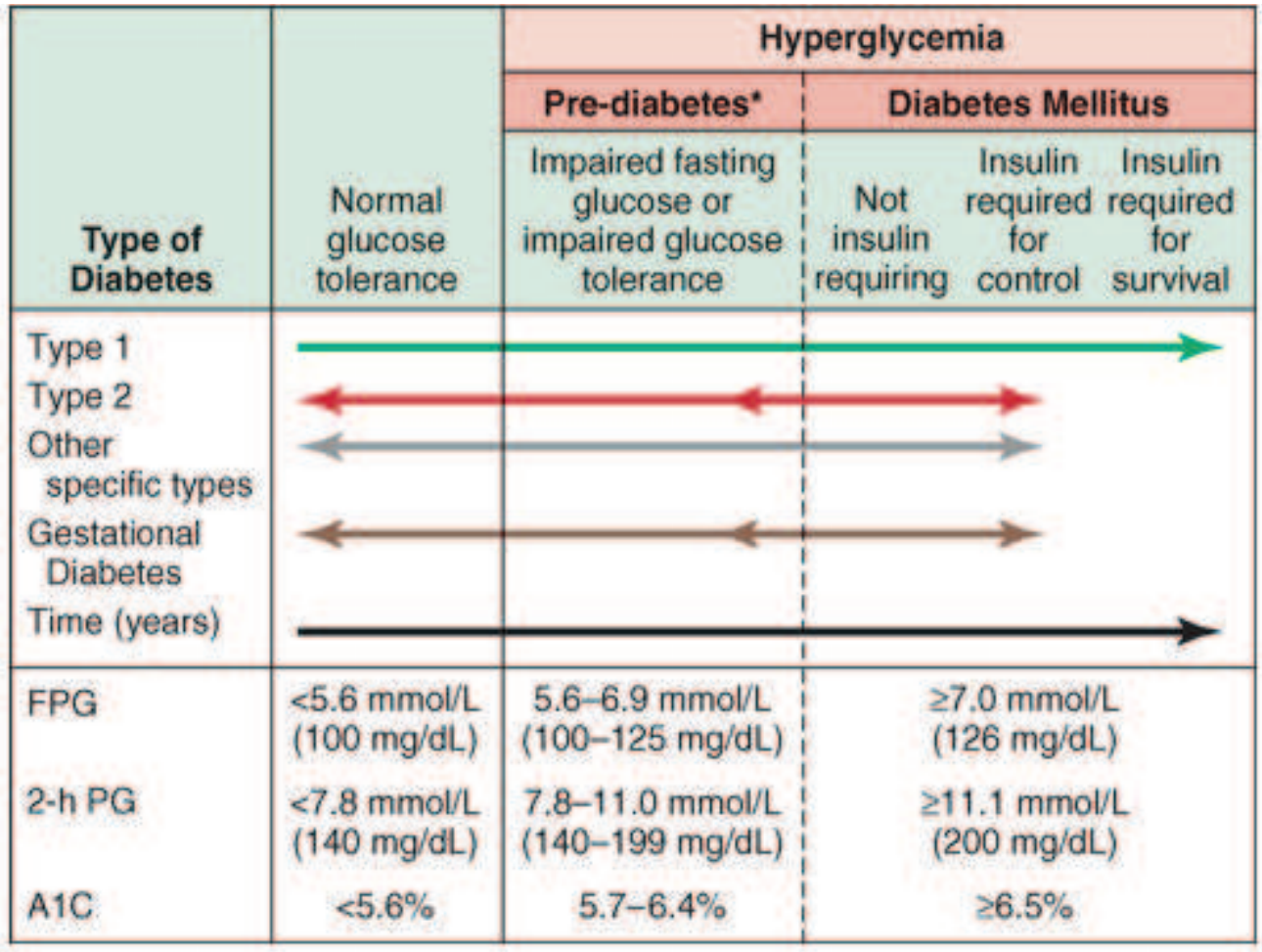
"Diabetes mellitus is a group of diseases characterised by insufficient production of insulin or by the failure to respond appropriately to insulin, resulting in hyperglycemia".

For the past two decades, the prevalence of diabetes has raised more dramatically to 285 million in 2012 when compared to 30 million cases in 1985.

According to the current trends, the estimation by International Diabetes Federation states that, by the year 2030 diabetes prevalence may increases upto 438 million. Compared to type 1, type 2 diabetes prevalence rises more rapidly which may be due to reduced physical activity, increasing obesity, industrialisation and it also increases with age of the individuals.

In the United States, the estimation of prevalence of diabetes during 2010 showed that 11.3% in individuals with >20 years age group, 0.2% in individuals aged < 20years, 26.9% in individuals aged >65years. In men and women the prevalence is similar in most age groups (11.8% in men and 10.8% in women, in individuals aged >20years). According to the worldwide estimation, by 2030 the diabetic individuals with age group 45-64 years will be more.

Eventhough much attention has been focused mainly on prevention and treatment of microvascular complications like nephropathy, neuropathy and retinopthy, the principal morbidity and driver of mortality in patients with diabetes are cardiovascular diseases (CVD) but also there is increased risk of cerebrovascular disease, heart failure and peripheral vascular diseases.



FPG – Fasting Plasma Glucose, 2-h PG – 2 Hour Plasma Glucose

TABLE 2: Criteria for diagnosis of Diabetes mellitus

II. 3. ANATOMY OF THE HEART

Most of the hollow organs of the body are made up of different types of muscular layers, but heart is entirely made up of a single muscle layer. In most hollow organs of the body muscle layers are composed of smooth and striated muscles but in the heart it is composed of specialised tissue cardiac muscle.

All muscle types functions by contraction, causing the muscle cells to shorten. Skeletal muscle cell acts voluntarily after getting signals from brain. The smooth muscles which surround the hollow organ acts involuntarily; they do not need a signal from brain. Cardiac muscle also acts involuntarily. So, cardiac and smooth muscles are functionally similar. But cardiac and skeletal muscles are anatomically resembles more closely. Both cardiac and skeletal muscles are striated.

Cardiac muscles have several unique features. Three types of cardiac muscle are present, which includes atrial muscle, ventricular muscle, and specialised excitatory and conductive muscle fibres. Like skeletal muscle, cardiac muscle also has actin and myosin filaments. During contraction these filaments lie side by side and slide along one another. Cardiac muscle acts as a syncytium. They contain intercalated discs, which are cell membranes that separate the individual cardiac muscle cells from one another.

After slight stretching cardiac muscle contracts more powerfully. When the ventricles are filled, beyond their normal capacity they are stretched. This results in most powerful contraction of ventricles and hence maximum amount of blood forced into the arteries with each stroke.

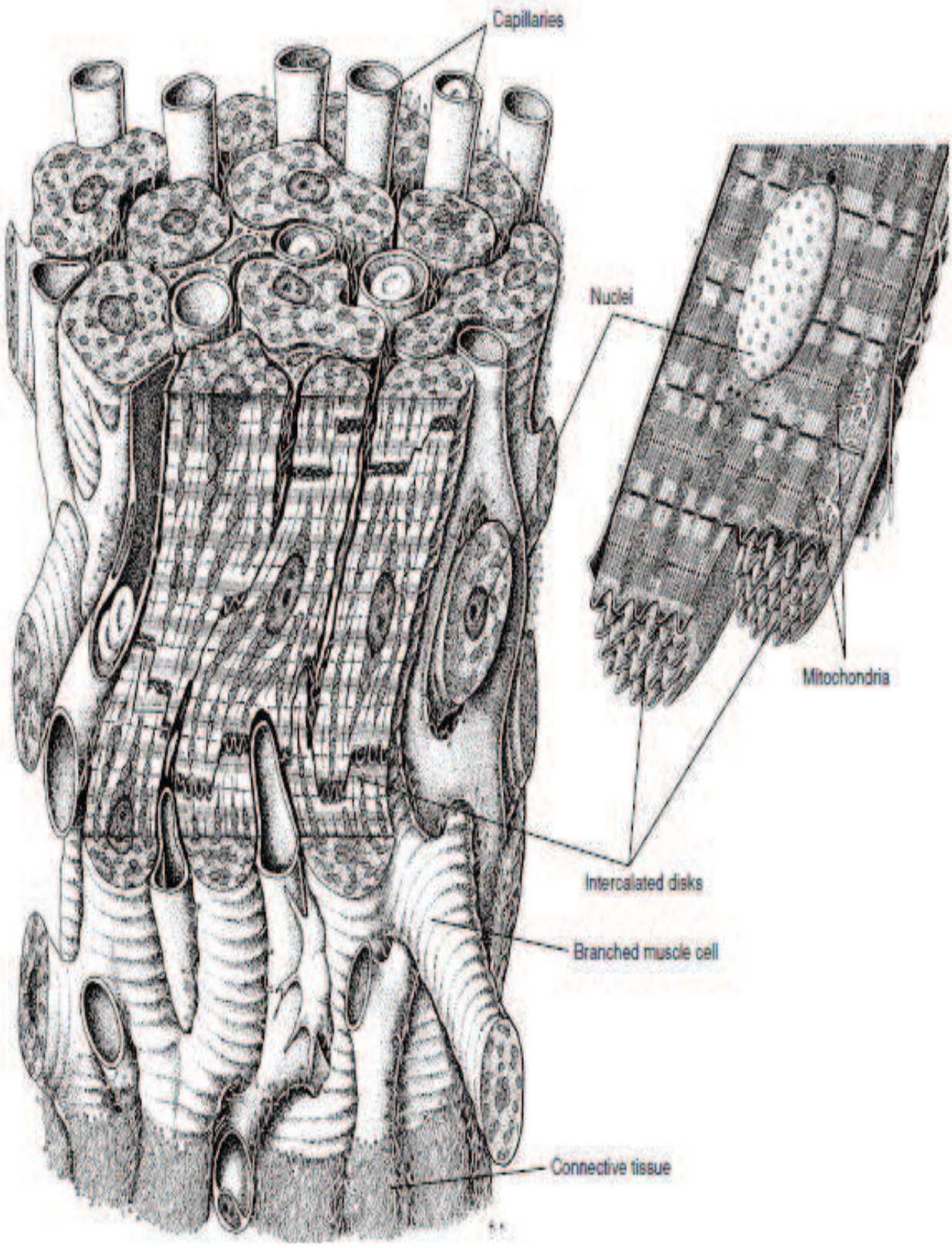


FIGURE 1: Structure of the Cardiac Muscle

The heart has four chambers, two atria and two ventricles. The atria receives blood from the veins which then contracts and pushes blood into the ventricle. Even if the atria fail to contract, most of the blood in atria will flow into the ventricle. Hence atria do not really have to work that hard. However the ventricles are the real work horses, since they must have sufficient power to push the blood away from the heart all the way to the last point of arterial tree.

The ventricular musculature is much thicker than the atrial musculature. The heart walls are made up of several spirally arranged muscle fibres, which push the blood from the ventricles during contraction. Heart valves present between the atria and ventricles allows unidirectional flow of blood.

The heart is composed of involuntary muscle which has self excitatory. Hence they can initiate contraction themselves.

Linzbach (1960)[1] studied that for every myocardial fibre there is one capillary in a ratio of 1:1. In hypertrophied heart this remains same. Thus capillary inadequacy is not a cause of hypoxia in a hypertrophied heart. The ratio between coronary artery and coronary ostial diameter decreases in marked hypertrophy, which restricts myocardial perfusion in hypertrophied heart.

Spann et al (1969)[2] had shown that in a non-failing hypertrophied heart, there is decrease in the development of tension and contraction in myocardial tissue which may ultimately leads to congestive cardiac failure later in due course of time.

Grove et al (1969)[3] showed that hyperplasia of myocardial cells occurs only in the embryonic and early postnatal growth.

Any subsequent myocardial enlargement in adult life can only be due to hypertrophy of the individual myocardial cells.

Benzak (1969)[4] showed experimentally when cardiac workload returns to normal, myocardial cells returns to their original size.

Meerson (1969)[5] had stated that compensatory hyper functioning of heart leading to hypertrophy occurs in response to abnormally increased workload.

Laks et al (1974)[6] showed that an increase in cell length as a result of adding more sarcomeres cause cardiac hypertrophy rather than the result of stretching of sarcomeres.

II. 3. a. Left ventricular hypertrophy:

Left ventricular hypertrophy is defined as "An abnormal increase in the mass of the left ventricle" which is an independent risk factor in predicting causes of cardiovascular morbidity and mortality like congestive heart failure, myocardial infarction and sudden cardiac death.

Pathologically LVH is defined as an "Increase in volume of cardiac myocytes"[7]. To normalise the wall stress, there occurs remodelling of the architecture of heart when there is hypertrophy of ventricles. The type of load imposed on the heart predicts the pattern of hypertrophy.

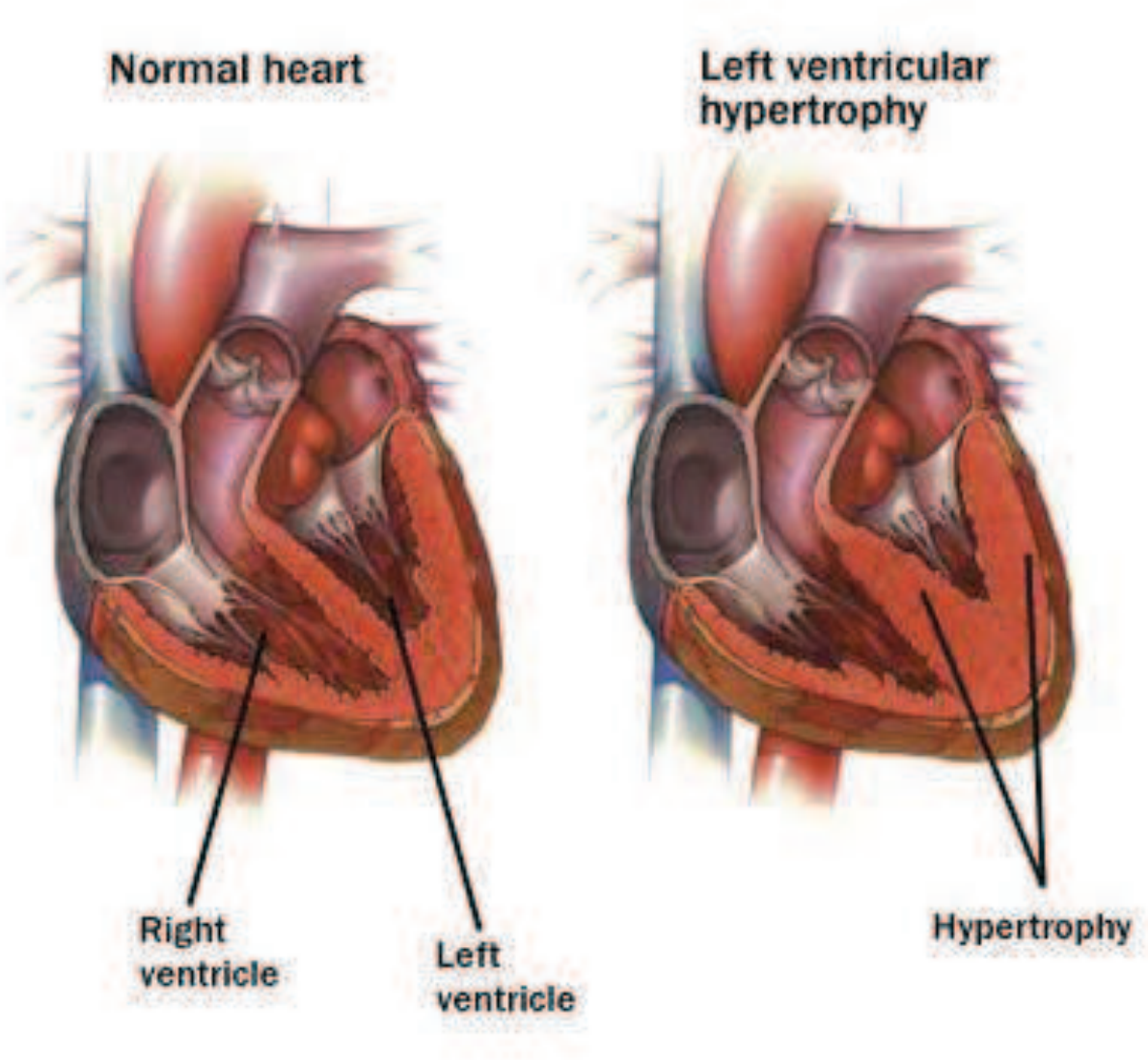


FIGURE 2: Pictorial Differentiation of Normal Heart and Left Ventricular Hypertrophy

Increased afterload causes an increase in systolic stress and also an increase in number of sarcomeres in parallel manner leading to increase in wall thickness with decrease in chamber size. It is termed as concentric hypertrophy[7]. It is seen in patients with pressure overload as in systemic hypertension.

Increased preload causes an increase in diastolic stress which causes sarcomeres to be added in series leading to increase in chamber size. This increases ratio of chamber size to wall thickness and this type of ventricular hypertrophy is termed as eccentric hypertrophy[7] which commonly occurs in case of volume overload of left ventricle like aortic regurgitation, mitral regurgitation. End stages of dilated cardiomyopathy are associated with eccentric hypertrophy. Clear distinction between concentric and eccentric hypertrophy can be made with a help of echocardiography.

In athletes, due to strenuous physical exercise, there occurs increased left ventricular mass with normal function. Pathological stress on the heart leading to left ventricular hypertrophy is associated with diverse consequences.

II. 4. PHYSIOLOGY OF CARDIAC CONTRACTION

II. 4. a. Myocardial Excitation Contraction coupling:

The myocardial tissue is made up of multiple fibres which in turn made up of multiple myofibrils. Each myofibril consists of subunits called as sarcomeres which are separated by horizontal lines called as Z lines which project into the sarcomeres and integrate with the thicker filament portion myosin.

Myosin acts as an adenosine triphosphatase by splitting adenosine triphosphate. Actin and myosin interacts and produces actomyosin which is a more potent ATPase when compared to myosin alone.

Troponin and tropomyosin are the two other proteins which inhibit the contraction of actomyosin but when calcium ions binds to troponin and tropomyosin, these proteins are unable to inhibit actomyosin and contraction occurs. Thus, calcium ion is an inotropic factor.

There are two distinct channel systems on sarcolemma which are the 'T' system and sarcoplasmic reticulum which is a storage site for calcium ions.

There is a high intracellular concentration of potassium (K+) and low calcium (ca2+) and sodium (Na+) concentration in the sarcolemma during diastole which is maintained by cell membrane by an active process and is enzyme dependent. When depolarisation occurs there is rapid entry of sodium into the cells during the first phase of action potential.

During the second phase calcium ions enters slowly into the cells, which can be blocked by magnesium ions and drugs like verapamil and acidosis, opposite effect is exerted by methyxanthine and catecholamine which increases calcium content of myocardial cell producing an inotropic effect. There is a relationship between intracellular calcium and sodium concentration. When sodium is extruded out of the cell, for every three sodium ions one extracellular calcium ion enters into the cell resulting in increased intracellular concentration.

II. 4. b. The Role Of Muscle Length:

The force of contraction depends on the initial muscle length. The sarcomere length of 2.2micrometers is associated with most powerful contraction. The two sets of sarcomere myofilaments are ideally situated at this

length to provide the greater area for their interaction. The thin filaments are entirely withdrawn from the A band and no tension develops when the sarcomere length is increased to 3.65micrometers. The capacity of force development also declines when the sarcomere is shorter than 2micrometer. Since the thin filaments bypass one another and thereby producing a double overlap, which leads to cause a reduction in the sensitivity of contractile sites.

The basis of the Frank starlings reaction (starlings law of heart) which states that "Within limits the augmentation of initial volume of the ventricle, which is function of the initial length of the cardiac muscle, results in an increase in the force of the ventricular contraction".

II. 4. c. The Force Velocity Curve:

The mechanical activity of all muscle depends on shortening and development of tension. There is inverse relationship between the velocity of shortening and magnitude of tension development within the muscle. There is alteration in the contractile activity of the myocardium under physiological conditions by changes in the inotropic states and changes in the resting muscle fibre length which shift the myocardial force velocity curve.

II. 4. d. Ventricular Wall Tension And The Laplace Relationship:

Afterload is the resistance against which the ventricles contract i.e., the arterial wall resistance, the peripheral vascular resistance, aortic impedance, the column of the blood mass in the aorta and the viscosity of the blood.

Ventricular myocardial pressure is related to the intraventricular pressure per centimetre square of surface upon which the pressure is exerted.

At the beginning of ventricular ejection and the time of peak systolic pressure, the wall tension begins to decrease and less than that at the onset of systole. The stimulus for myocardial hypertrophy is believed to be this increased tension in dilated hearts.

There is increased ventricular performance several beats after aortic pressure raise is the additional influences of alteration in afterload. Some studies state that this phenomenon can be due to recovery from transient subendocardial ischemia which is caused by sudden alteration in arterial pressure.

II. 4. e. Contractility And The Ionotropic State:

The change in the inotropic state (contractility) of the muscle which is independent of the change in afterload or preload (fibre length) alters the myocardial function is the third major mechanism. This indicates that when there is augmentation of contraction, at any given length for any given load the myocardium shortens faster and generates force.

II. 4. f. Heart Rate (Bow Ditch Effect):

The heart rate or the frequency of cardiac contraction is the fourth major determinant of cardiac function. Because of this mechanism, during the periods of exercise or increased demand there is increased cardiac output. Increased contraction and relaxation also occurs due to increased heart rate which

improves diastolic performance. The TREPPE, STAIRCASE PHENOMENON or BOWDITCH EFFECT is the systemic force-interval relationship.

The negative or reverse staircase phenomenon or WOODWORTH PHENOMENON is the “recuperative effect of a long pause” upon the strength of contraction.

II. 4. g. Other Factors:

Atrial function, ventricular diastolic dysfunction, adequacy of ventricular contraction, hormonal control, nervous control etc., are the other factors that influence ventricular contraction.

II. 5. LEFT VENTRICULAR HYPERTROPHY PATTERNS AND PATHOGENESIS:

II. 5. a. Patterns of Left Ventricular Hypertrophy:

One of the principles involved in the heart’s compensatory activity for increased load is the development of left ventricular hypertrophy. In both the volume and pressure loaded conditions, there is approximately equally increased left ventricular systolic stress, mass and diastolic pressure. In volume loaded ventricles, the wall thickness was increased substantially. The latter was just enough to counter balance the increased radius so that in volume overloaded conditions, there is normal wall thickness to radius ratio, while there was disproportionate thickening of ventricular wall. Thus the systolic stress is maintained relatively unchanged in compensated patients. The compensatory

mechanism for increased load is hypertrophy, which adversely causes slowing relaxation and increasing stiffness.

This stiffness can be due to

1. An increased intrinsic stiffness of myocardium (due to myocardial ischemia, fibrosis, amyloid infiltration).
2. Without alteration of intrinsic ventricular myocardial thickness, there is an increased ventricular wall thickness and mass.
3. Can be due to combination of these two mechanisms.

II. 5. b. Morphology of cardiac hypertrophy:

II. 5. b. i. Early stage:

In this stage, myofibrils and mitochondria will increase in number, which is followed by enlargement of mitochondria with nuclei and increasing length of muscle cells with preservation of cellular organisation.

II. 5. b. ii. Advanced stage:

In this stage also the specific organelles size and numbers are increased. Along with this there is occurrence of subtle changes in cellular organisation due to localised cell areas have irregular addition of new contractile elements.

II. 5. b. iii. Long standing Hypertrophy:

Here the nuclei are markedly enlarged and high lobulation of membranes with breakdown of normal Z- band registration. The cellular organisation is more obviously disrupted in this stage.

II. 5. b. iv. Late stage of hypertrophy:

There is marked disruption of Z bands with loss of contractile elements.

The changes include:

- Severe disruption of the parallel arrangement of sarcomere
- Fibrous tissue deposition
- T-tubules showed dilatation and increased tortuosity.

II. 5. c. Chronic mechanical overload of Heart and changes in membrane proteins:

Mechanical origin of cardiac hypertrophy causes alteration of number and density of membrane proteins in an equivocal way. There are two distinct groups of protein distinguished.

- The calcium^{2+}-ATPase of the sarcoplasmic reticulum, the low affinity isoform of the $Na^{+}k^{+}$ATPase and beta one adrenergic and muscarinic receptors form one group of membrane proteins. Because of hypertrophic process not involved in activation of protein synthesis in this group, their changes in number per cell do not occur. The adaptive slowing down of relaxation is reflected by the decreasing density of ca^{++}ATPase of the sarcoplasmic reticulum, whereas the beta receptors down regulation have some protective role.
- The calcium channels and also by one isoform of $Na^{+}k^{+}$ATPase forms another group of membrane proteins. Because of this group of proteins activated proportionately by the degree of hypertrophy, their number per cell

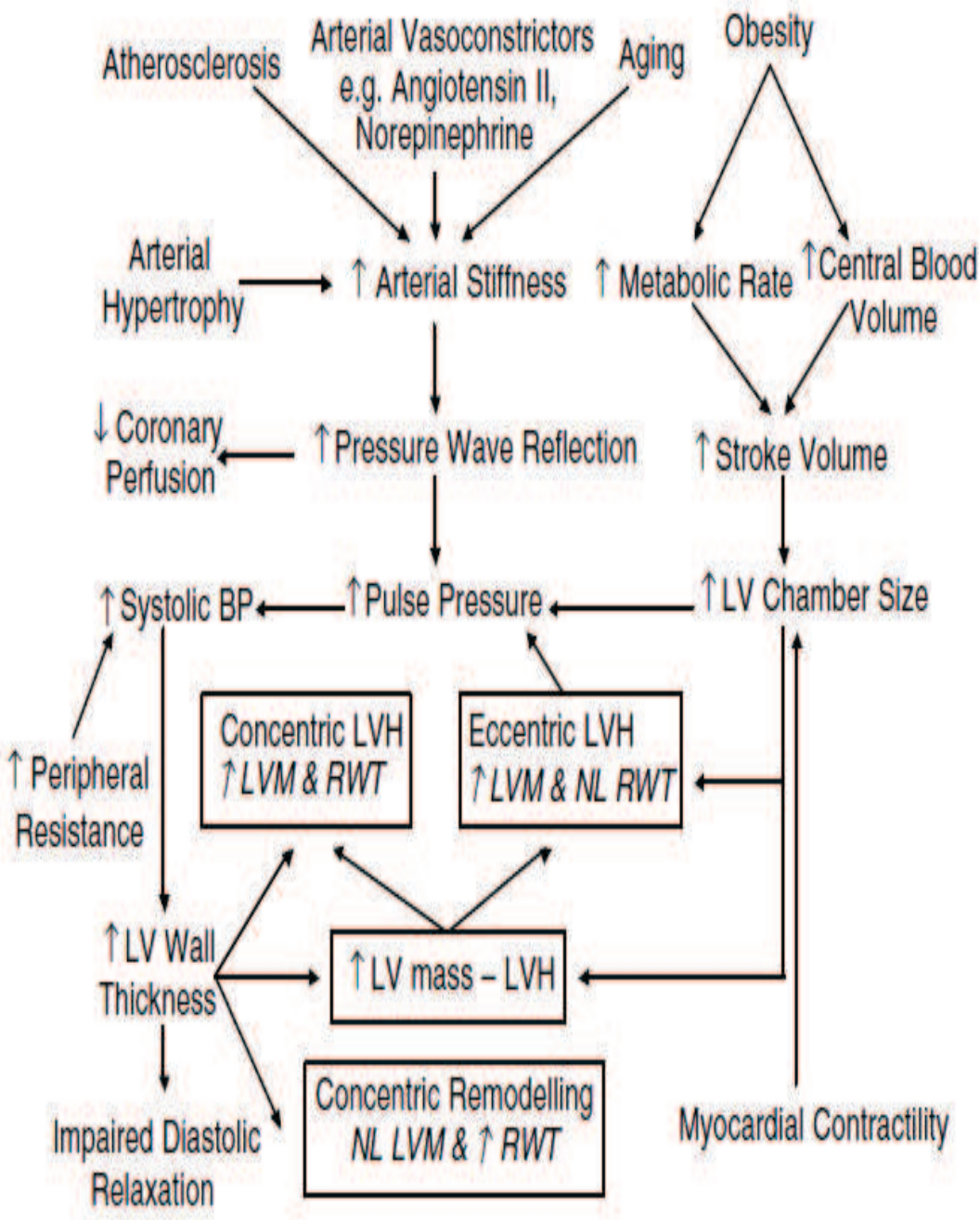
increases. The chronic mechanical overload induces the gene coding for these proteins.

In volume overloaded conditions, activation of matrix metallo-proteineases (gelatinase, collagenase and stromalysins) [79, 80, 81] produces tissue inhibitors down-regulation and thereby causing chamber expansion by changing collagen matrix.

II. 5. d. Hemodynamic Stimuli In The Development Of Left Ventricular Hypertrophy [89]:

Arterial vasoconstrictors like angiotensin II and norepinephrine increases arterial stiffness. This increases pulse pressure which in turn further increases systolic blood pressure. Chronic high blood presssure leads on to produce left ventricular hypertrophy and thereby increases left ventricular mass. This increased LV mass produces remodelling effect on left ventricle and produces diastolic dysfunction.

Other factors like atherosclerosis also increases LV mass by the same way like hypertension. . In obesity, increased metabolic rate increases stroke volume which in turn produces volume overloaded hypertrophic changes on heart which is called as ecccentric hypertrophy.



LV- Left ventricle, **LVH** – Left ventricular hypertrophy, **NL-** Normal, **LVM-** Left ventricular mass, **RWT-** Regional wall thickness

Figure 3: Hemodynamic Stimuli In The Development Of Left Ventricular Hypertrophy [89]

II. 6. ETIOPATHOGENESIS OF LEFT VENTRICULAR HYPERTROPHY IN HYPERTENSION:

The preferential synthesis of mitochondria is the first cellular change that occurs after the stimulus for hypertrophy, presumably the sufficient adenosine triphosphate provided by expanded myocardial mass which helps to meet the energy demands of the hypertrophied cell. Then the myofibril mass will increase later. This can taken place only when there is depression of the DNA in the nucleus of myocytes, which allows DNA replication to occur.

The stimuli for left ventricular hypertrophy includes

- ATP depletion.
- Because of sustained increase in preload and afterload there is stretching of myocytes do occurs (sympathetic nervous system).
- Due to wear and tear mechanism there is accumulation of products of cell degeneration occurs.
- Humoral factors for stimuli include rennin-angiotensin, thyroid hormones, growth hormones, epinephrine, etc.

L.H Missault et al (2002) [60] studies the relationship between left ventricular mass and blood pressure. There is a higher correlation when systolic blood pressure or night-time blood pressure is considered. In conclusion the relationship between blood pressure and left ventricular mass is low once the hypertension is treated. It is found that night-time blood pressure dippers have

better prognosis than non-dippers and hence night-time blood pressure better correlates with left ventricular mass than day-time blood pressure [61].

Hond et al (2003) [63] studied the relationship between systolic blood pressure and left ventricular mass using baseline left ventricular mass and different types of blood pressure like 24-hour ambulatory blood pressure, day-time blood pressure, office-blood pressure and night-time blood pressure. There is a strong association of echocardiographic mean wall thickness with ambulatory blood pressure than with office blood pressure.

II 6 a. Sympathetic Nervous System Influence On Myocardial Hypertrophy:

Sympathetic innervation of the heart mediates the increased heart rate and myocardial contractility due to pressure and volume loaded conditions. Norepinephrine is described as a myocardial hypertrophy hormone by laks[6]. Through alpha receptors norepinephrine stimulates the growth of isolated cultured myocardial cells as shown by Samson. Diuretics stimulates sympathetic nervous system despite causing marked decrease in blood pressure, diuretics did not lead to regression of cardiac hypertrophy. This explains the failure of diuretic therapy produces reversal of hypertrophy.

Administration of methyldopa leads to decrease in norepinephrine in circulation, which leads to reduction of left ventricular mass without reduction in blood pressure.

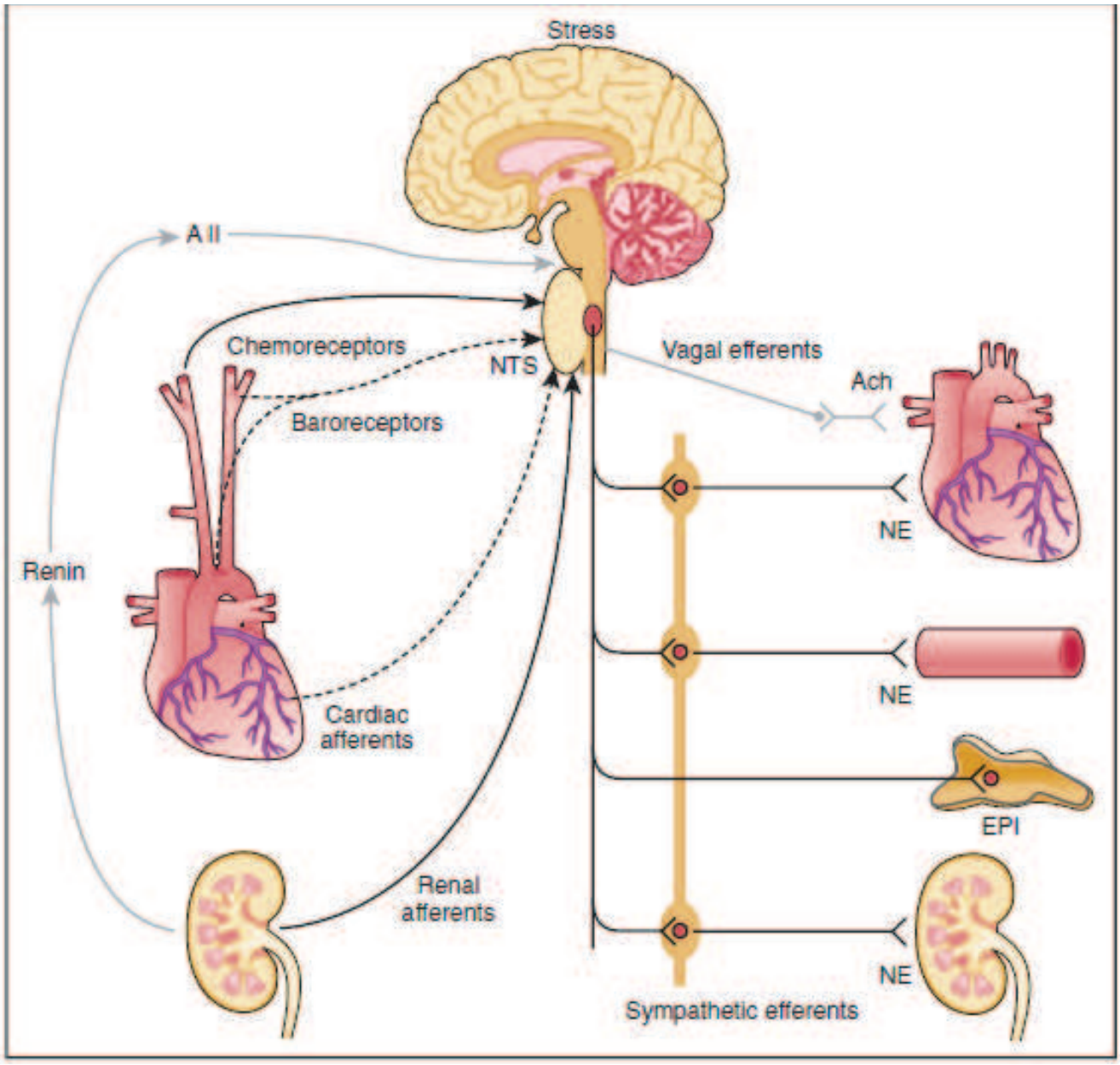


FIGURE 4: Sympathetic Nervous System Influence On LVH

A solid arrow represents the excitatory neural influences and inhibitory neural influences on sympathetic outflow of heart, kidneys and peripheral vasculature are represented by dotted arrows. AII –Angiotensin II, EPI – epinephrine, NE – Norepinephrine, NTS – Nucleus tractus solitarius, Ach – Acetylcholine.

Whereas treatment with alpha blockers (clonidine) leads to reduction of left ventricular mass and associated decrease in blood pressure

Similarly beta adrenergic antagonists also cause reduction of left ventricular hypertrophy. However after 12 months of treatment with beta blockers (metoprolol) J. Wikman- coffelt et al[8] demonstrated very protracted regression of left ventricular hypertrophy.

II. 6. b. Genetic Determination Of Myocardial Hypertrophy:

In 14% of hypertensive patients, there is asymmetrical septal hypertrophy.

This may be due to the following reasons:

- Ventricular septum hypertrophies earlier in response to increased systemic pressure load even before the hypertrophy of anterior or posterior wall on account of its longer radius of curvature.
- The concentration of catecholamines is higher in ventricular septum which account for the higher prevalence of septal hypertrophy on hypertension.

Since essential hypertension is genetically determined, the response of heart to hypertrophy is also being genetically determined. There occurs an individually varying genetic component in all cases of hypertensive cardiac hypertrophy. This explains the fact that response to antihypertensive treatment varies in different patients.

II. 6. c. Endothelial Cell Dysfunction:

The relaxing factors such as prostacyclin (PGI2), nitric oxide (NO) and endothelium derived hyperpolarizing factors are released when specific receptors (organ circles) on endothelial membrane by blood and platelet derived substances. Contracting factors like angiotensin II, endothelin I, prostaglandin H2 (PGH2), and thromboxane A2 (TXA2) are also released from the endothelial membrane.

The major defence against hypertension is constituted by the endothelial lining of blood vessels which is an important factor which determines the vascular health. Dysfunctional endothelium is characterised by an imbalance between endothelium derived contracting and relaxing factors, which causes increased release of endothelium derived constricting, prothrombotic, proinflammatory and growth factors like transforming growth factor beta (TGF–β), thromboxane and endothelin. There is decreased release of endothelium derived relaxing factors like endothelium derived hyperpolarizing factor and nitric oxide.

Francesco et al (1999) [65, 66] showed that both endothelial dysfunction and left ventricular hypertrophy forms the background for the development of hypertension. This study showed that there is a inverse relationship between endothelium dependent vasodilating agent acetylcholine and echocardiographic left ventricular mass in hypertensive patients.

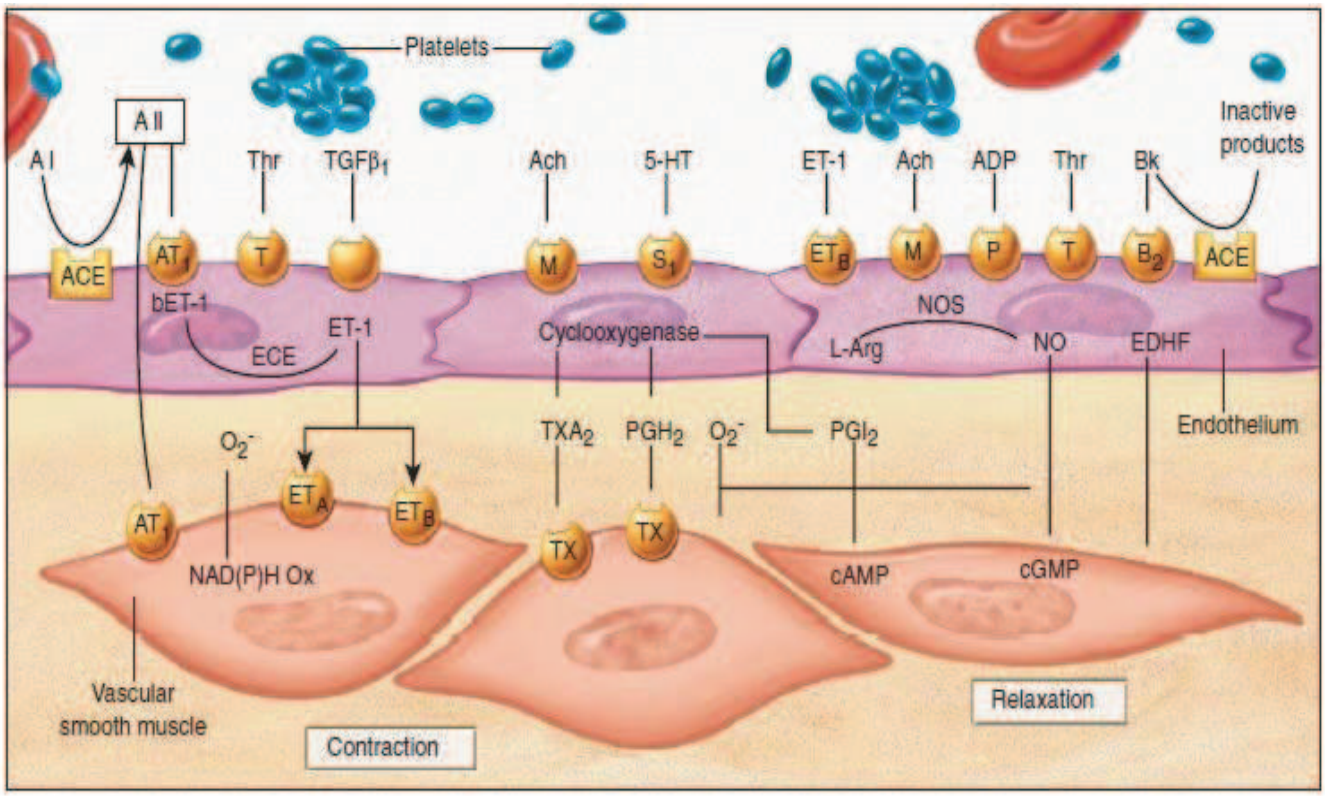


FIGURE 5: Endothelium Derived Relaxing And Constricting Factors [104]

The activation of specific receptors (orange circles) on the endothelial membrane by various blood and platelet-derived substances causes release of relaxing factors like prostacyclin (PGI2), nitric oxide (NO) and endothelium – derived hyperpolarizing factor (EDHF). There is also release of contracting factors like endothelin (ET-1), thromboxane A2 (TXA2), angiotensin (AII) as well as prostaglandin H2 (PGH2), 5-HT – serotonin, ACE - angiotensin-converting enzyme, Bk- bradykinin, L-Arg - l-arginine, ECE - endothelin-converting enzyme, NOS - nitric oxide synthase, TGFβ1- Transforming growth factor- β1, O2− = superoxide, Thr – Thrombin. .

II. 6. d. Vascular Remodelling:

Overtime elevated blood pressure, neurohormonal activation and endothelial dysfunction causes remodelling of blood vessels [95,96]. In small and large arteries the hallmark of hypertensive remodelling is an increase in the media thickness compared to lumen diameter (ratio of media-lumen is increased).

Vasoconstriction is the initial effect of remodelling in the small arteries, which normalises wall stress and thereby averts the trophic response. A smaller lumen diameter was surrounded by rearrangement of normal smooth muscle cells; this process is called as inward eutrophic remodelling. Without alteration of medial cross sectional area, there is increase in media to lumen ratio.

The hemodynamic hallmark of diastolic hypertension is "In the peripheral circulation, by decreasing lumen diameter systemic vascular resistance is increased by inward eutrophic remodelling".

In large arteries the remodelling process is characterised by, triggering expression of hypertrophic genes which in turn increases medial thickness and media to lumen ratio.

This hypertrophic remodelling is characterised by increased in size of vascular smooth muscle cells and also extracellular matrix proteins such as collagen and fibronectin accumulation which is caused by TGF-β activation. This in turn produces stiffness of large arteries which is the hemodynamic hallmark of isolated systolic hypertension.

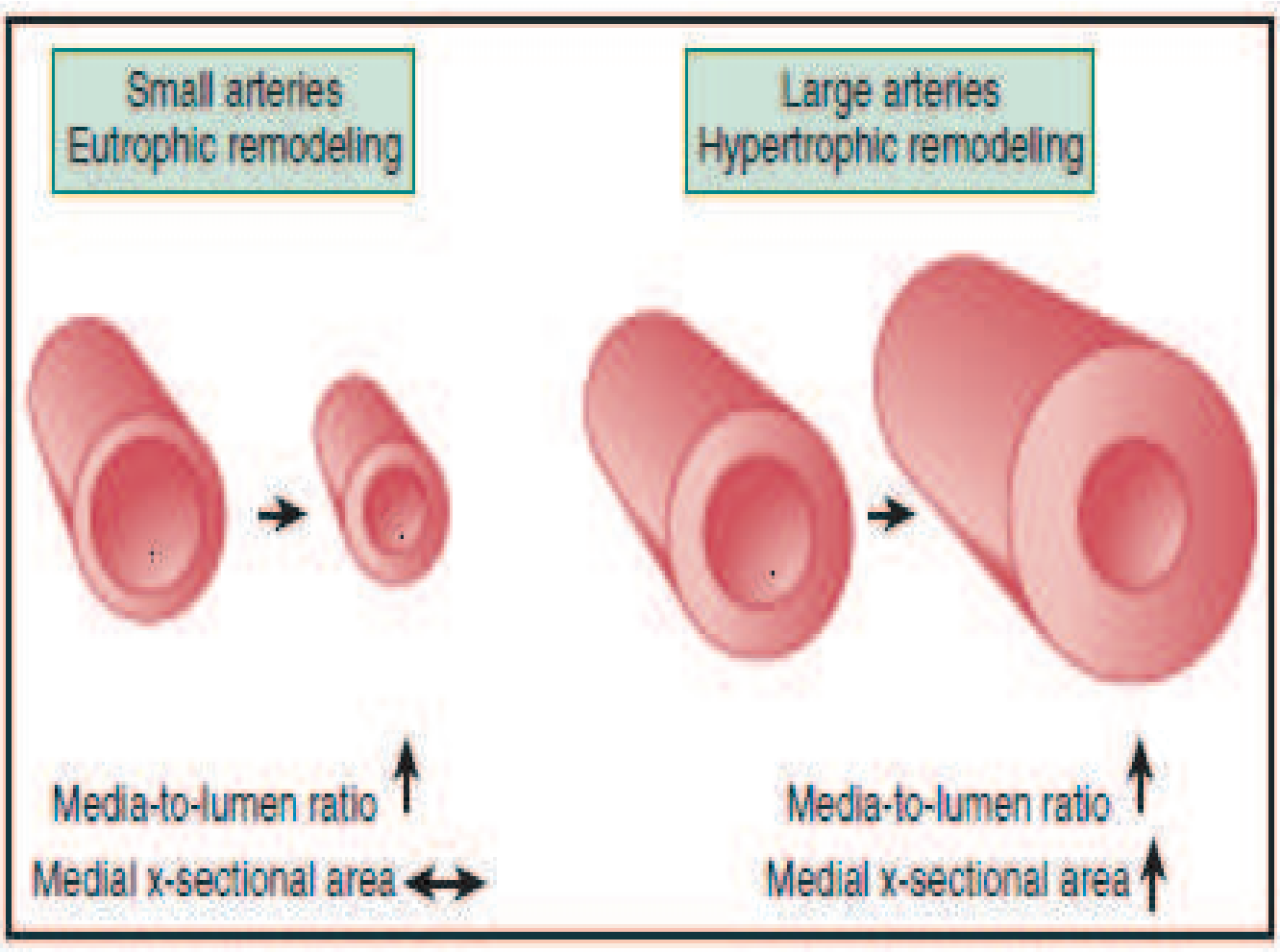


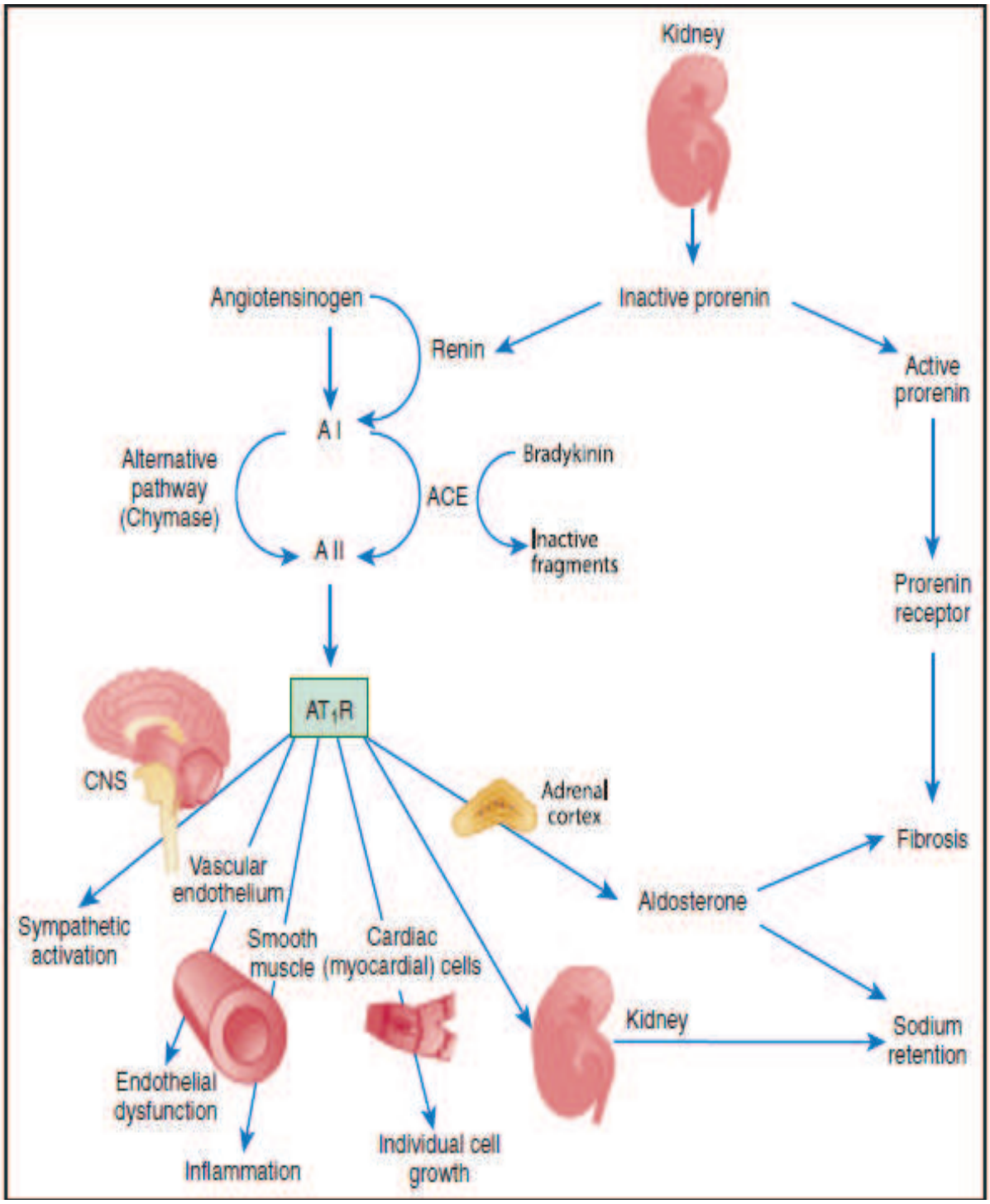
FIGURE 6: Vascular Remodelling Of Small And Large Arteries In Hypertension [103]

Tunica media, tunica adventitia, tunica intermedia are showed in this diagram by cross section of arteries.

II. 6. e. Hormonal Mechanisms Involved In Hypertension:

II. 6. e. i. Renin-Angiotensin Aldosterone System:

The one of the most important mechanism which involved in endothelial cell dysfunction is the renin – angiotensin – aldosterone system. Because of activation of this mechanism vascular remodelling and hypertension occurs.



AI – Angiotensin I, AII- Angiotensin II, ACE – Angiotensin converting enzyme, AT1R – Angiotensin 1Receptor.

FIGURE 7: Renin-Angiotensin Systen Activation In HT

Renin is a protease, secreted by renal juxtaglomerular cells, which cleaves the angiotensinogen to angiotensin I, this further produces angiotensin II by angiotensin converting enzyme.

Angiotensin converting enzyme is most abundantly present in the lungs and also in the systemic vasculature and (tissue ACE) and heart. The alternative pathway for conversion of angiotensin II from angiotensin I is by a serine protease chymase which is present in the heart and systemic arteries. The numerous cellular processes that are involved in hypertension and its end organ damage is produced by the interaction between G- protein coupled angiotensin I receptors and angiotensin II. The cellular processes involved in hypertension includes vasoconstriction, vascular inflammation, generation of reactive oxygen species, the production of aldosterone, the principal mineralocorticoid and vascular and cardiac remodelling.

There is increasing evidence that the damage of vascular health thereby causing hypertension is due to activation of multiple signalling pathways by angiotensin II, aldosterone and even renin and prorenin.

II. 6. e. ii. Effect of Renin-Angiotensin system on left ventricular hypertrophy:

During treatment with angiotensin converting enzyme inhibitors there is regression of left ventricular hypertrophy. This shows that angiotensin II has a strong role in causing left ventricular hypertrophy [97, 98]. Angiotensin II stimulates biosynthesis of myocardial protein has been reported by sahn et al[9].

Through angiotensin 1 receptor, angiotensin II induces molecular events and thereby producing hypertrophy [82, 83, 84, 85, 86].

Sympathetic tone is enhanced by angiotensin by its central and peripheral action on autonomic nervous system and thereby stimulates the adrenal medulla to secrete catecholamines.

Secondarily this tropic action of angiotensin II is mediated by catecholamines. This is proved by the fact that special angiotensin analogues which do not release catecholamines have no effect on myocardial hypertrophy.

II. 6. e. iii. Receptor mediated actions of Angiotensin II:

There are two types of angiotensin receptors. Angiotensin I receptor is widely present in the kidney, heart, liver, adrenals, vasculature and brain. Most of the hypertensive actions of angiotensin II occur due to the activation of Angiotensin I Receptor [100, 101]. The angiotensin I receptor activation leads on to produce vasoconstriction, cell growth and proliferation, aldosterone release, activation of sympathetic system, and inhibition of renin release which further produces cardiac hypertrophy.

The activation of angiotensin II receptor produces opposite actions of angiotensin I and thereby inhibits cardiac muscle hypertrophy.

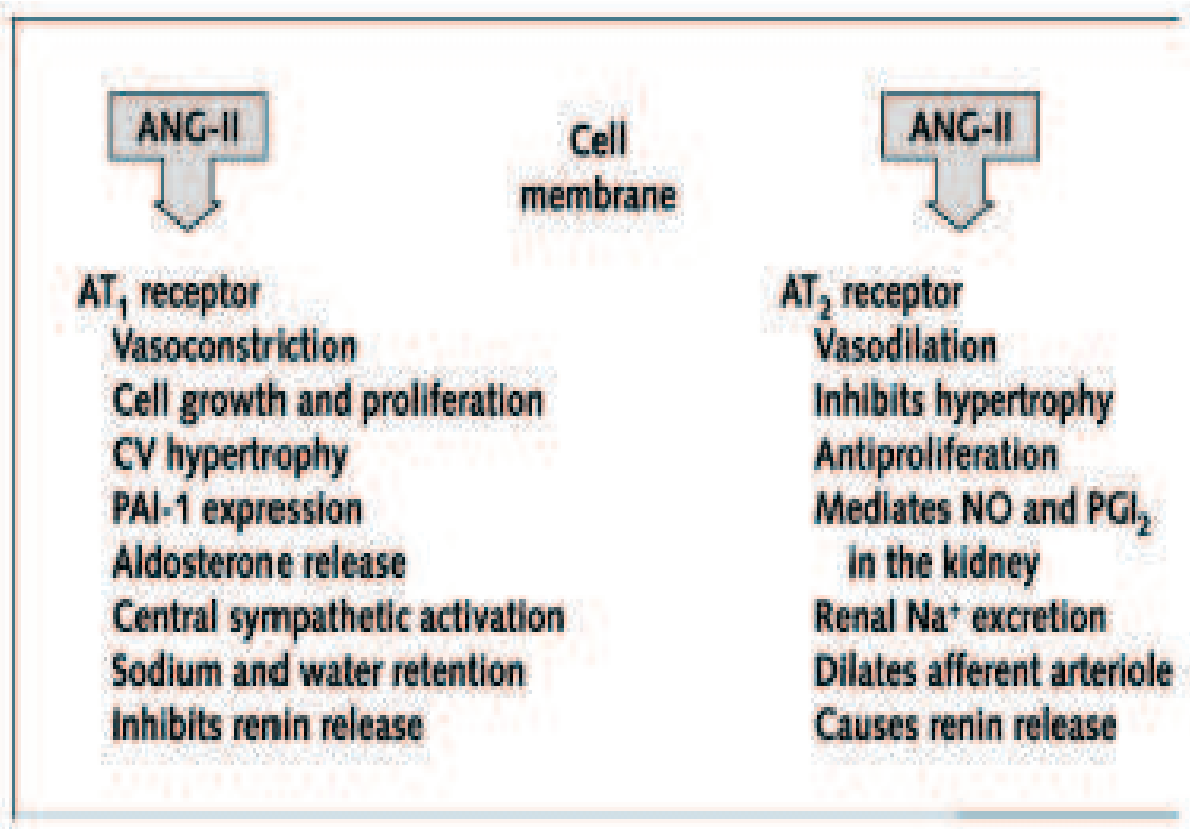
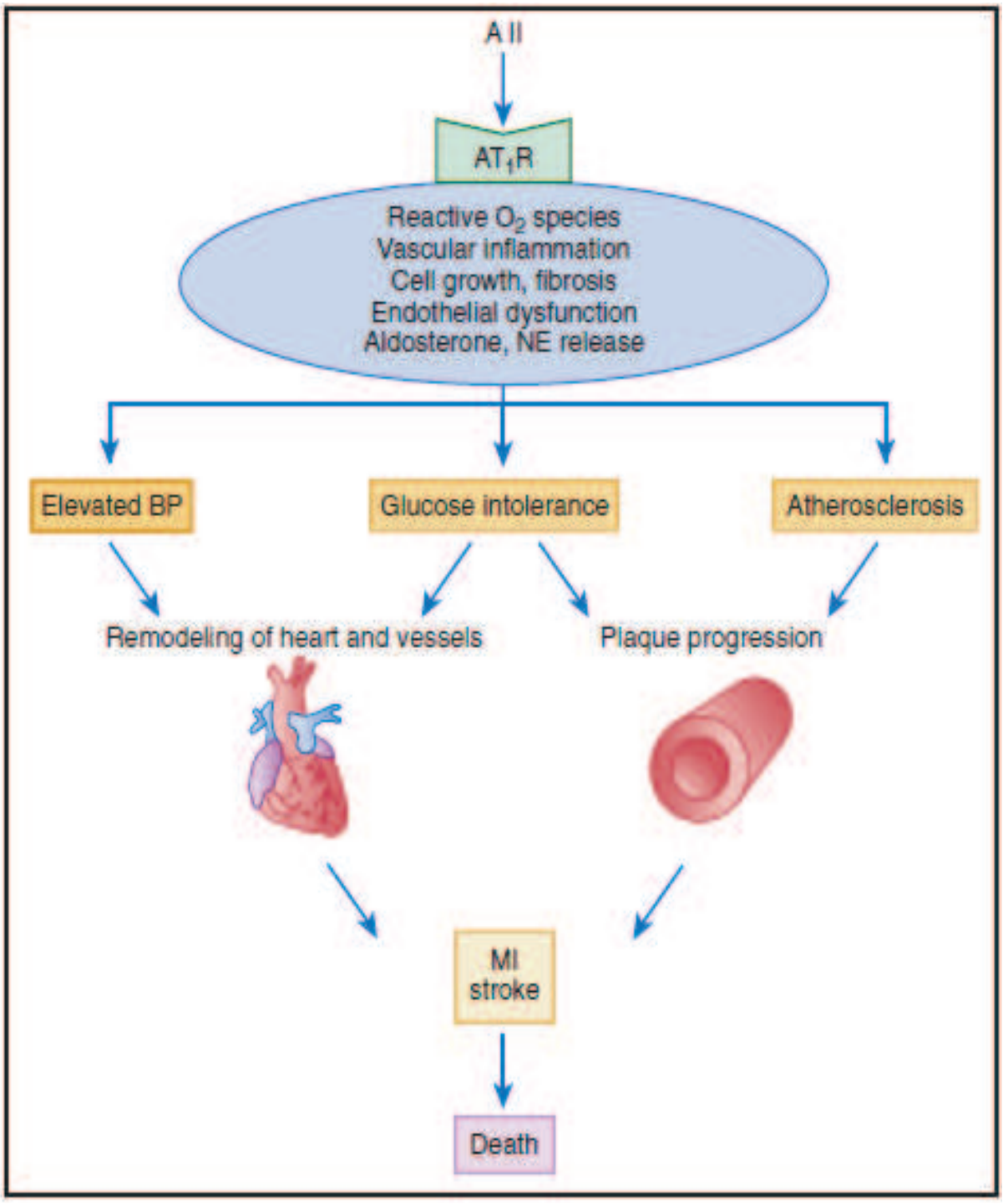


FIGURE 8: Opposite Actions Of Angiotensin II Receptors (AT1, AT2) [99]

PAI-1 – Plasminogen Activator Inhibitor-1; NO- nitric oxide; PGI2- Prostaglandin I 2 (Prostacyclin);

The enhanced angiotensin I receptor mediated signalling pathway, explains the mechanism for coexisting occurrence of high blood pressure with atherosclerosis and insulin resistance and this constitutes the therapeutic target for attenuating the progression of cardiovascular disease from vascular remodelling by formation of atherosclerotic plaque to myocardial infarction (MI), stroke and death.



AII – Angiotensin II, MI – Myocardial infarction, NE – Norepinephrine.

FIGURE 9: Cardiovascular Disease Progression By Angiotensin Receptor 1 Mediated (AT1 R) Signalling

II. 6. f. Reduced Coronary Reserve In Systemic Hypertension And Blood Rheology:

Hgn poiseuille law describes the relation between blood pressure, blood flow and blood viscosity. There is direct relationship between the blood flow to radius of the vessel and pressure gradient and inverse relationship between vessel length and blood viscosity.

The mechanisms involved in increase of plasma viscosity by arterial hypertension include:

- Because of transcapillary shift of fluid into interstitial space due to hydrostatic pressure there is reduction of intravascular volume.
- Due to activated interleukins, there is synthesis of fibrinogen degradation products in the precapillary vessels which increases synthesis of fibrinogen and other acute phase proteins from liver.

So the occurrence of arterial hypertension such as left ventricular hypertrophy and coronary vascular reserve reduction due to the following mechanisms:

- Inadequacy of vascular capacity.
- Secondary to medial hypertrophy, there is reduction of arteriolar diameter.
- The distensability of the pre-capillary resistance vessels has been reduced.
- Increased vasomotor tone like some functional alterations occurs.
- Blood fluidity has been impaired.

II. 7. INFLUENCE OF DIABETES MELLITUS ON LEFT VENTRICULAR MASS:

II. 7. a. Pathophysiologic changes Diabetes Mellitus:

Type 2 Diabetes Mellitus is characterised by insulin secretion impairment, insulin resistance, increased hepatic glucose production and fat metabolism abnormalities. The common factor in type 2 diabetes is obesity, particularly visceral or central which is evidenced by hip-waist ratio (≥ 80% are obese). Despite insulin resistance, the glucose tolerance remains normal in early stages of the disease, which is due to the development of compensatory mechanism of beta cells by increasing insulin output.

In certain individuals, the pancreatic islets are unable to sustain the hyperinsulinemic state due to the progression of insulin resistance and compensatory hyperinsulinemia. Following elevations of postprandial glucose, Impaired Glucose Tolerance (IGT) develops. A further decreased insulin secretion and increased production of hepatic glucose lead on to overt diabetes mellitus with high fasting blood sugar. Finally beta cell failure occurs.

II. 7. b. Background of left ventricular hypertrophy in diabetes:

Left ventricular hypertrophy is a frequent finding in diabetic individuals. It defines the powerful marker of poor prognosis of cardiovascular diseases including Americans and Africans. This left ventricular structure alteration not

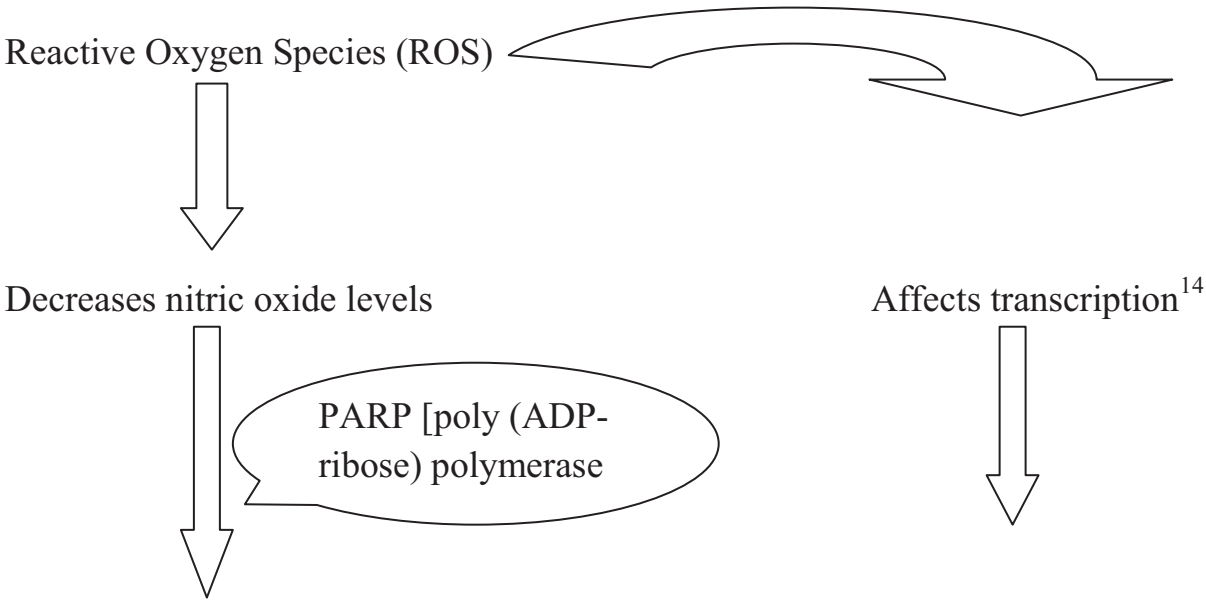
only related to diabetes but also related to hypertension, central obesity, obesity, aging, salt intake, dyslipidemia and physical inactivity. Among these hyperglycemia or hyperinsulinemia per se largely associated with left ventricular structural abnormalities. Individuals with type 2 diabetes mellitus echocardiographic left ventricular hypertrophy are usually associated with susceptibility to increased albuminuria[10] and atherothrombosis[11], which is a marker of endothelial dysfunction and microangiopathy[12]. This explains the pathologic link between inflammation and left ventricular hypertrophy.

II. 7. c. Mechanisms involved in increased left ventricular mass in Diabetes Mellitus:

The changes in gene expression, endothelial function, myocytes growth, myocardial substrate utilisation and myocardial compliance in the heart are due to alteration of downstream transcription factors which is induced by hyperglycemia, increased reactive oxygen species (ROS) and hyperlipidemia.

II. 7. c. i. Hyperglycemia:

The excess production of advanced glycation end-products (AGEs) is the principal abnormality which deactivates nitric oxide (NO) and impairs coronary vasodilatation. Prolonged hyperglycemia causes increased formation of mitochondrial reactive oxygen species (ROS) [13], which produces the following effects on the heart.



This advanced glycogen end-products related reactive oxygen species formation [(15)] also produces deposition of collagen in the myocardium and then myocardial fibrosis [(16,17)]. Metallothionein is a potent antioxidant which helps to prevent the formation of left ventricular hypertrophy [(18)] thereby increased left ventricular mass.

Hyperglycemia itself can promotes the left ventricular hypertrophy with involvement of transforming growth factor beta 1by synthesis of collagen by cardiac fibroblasts through the phosphatidylinositol 3- kinase and protein kinase C (PKC)[(19)] beta pathways.

II. 7. c. ii. Effect of Insulin on left ventricular hypertrophy:

Insulin acts as a growth hormone for cardiac myocytes through the protein kinase C (PKC) and/or extracellular signal regulated kinase (ERK) pathways.

Insulin also interacts with the neurohormonal system

⇩

Activates the sympathetic nervous system [20]

⇩

Increases the pressor response to angiotensin II

⇩

Increases the stimulating effects of angiotensin II on extracellular signal regulated kinase (ERK) pathways [21]

⇩

Cellular proliferation and extracellular matrix deposition

II. 7. c. iii. Fatty acids in Diabetes and Left ventricular mass:

Increased fatty acids in diabetes individuals and also increased dependence of diabetic myocardium on fatty acids produce several major cellular perturbances. So there is increased beta oxidation and accumulation of long -chain acyl carnitines in the mitochondria producing uncoupling of oxidative phosphorylation [22]. Inhibition of pyruvate dehydrogenase by enhanced fatty oxidation produces decreased glucose and pyruvate utilisation. This pyruvate oxidation is activated by Peroxisome-proliferator-activated receptor (PPAR) [23]. The net result is an excess production of glycolytic intermediates and also increased ceramide synthesis which leads on to apoptosis. PPAR- $\propto$ and - γ agonist prevents this apoptosis [23]. Thus impaired pyruvate oxidation, glycolysis and lactate uptake and also a fatty acid

dependence as a source of acetyl CoA can lead on to a perturbation of myocardial bioenergetics and contraction – relaxation coupling [24].

II. 7. c. iv. Renin – Angiotensin System influence in Diabetic Left Ventricular Hypertrophy:

In diabetes individuals, even minimal changes in myocardial loading up-regulates the renin-angiotensin system [25]. Insulin-like growth factor-1 (IGF-1) down-regulates the p53 DNA binding and thereby causes reduction in the transcription of angiotensinogen and also reduces angiotensin II levels results in reduction of cardiomyocyte apoptosis [26]. So insulin like growth factor-1 gene over expression down regulates the renin-angiotensin system and thereby inhibits the development of left ventricular hypertrophy [27, 28]. Cardiac hypertrophy produced by chronic activation of renin angiotensin activation system. This RAAS system in turn produces cellular alterations by angiotensin II and thereby producing cardiac hypertrophy [90, 91, 92, 93, 94].

II. 7. c. v. Aldosterone induced increased left ventricular mass:

Aldosterone and angiotensin II can increases LV mass by excessive accumulation of collagen and enhanced fibroblast proliferation in vivo [29,30,31,32]. In a dose dependant manner aldosterone and angiotensin II also enhance the human cardiac fibroblast to synthesize collagen in vitro [33, 34].

In a diabetic patient, because of alterations in the microvasculature, diffuse fibrosis has been noticed throughout the myocardium. Analysis of cardiac tissue in a diabetic patients showed that there is accumulation of collagen in interstitial and focal perivascular area which indicates the fibrosis [35]. In a diabetic patient, hyperglycemias dysregulates renin-angiotensin - aldosterone system thereby produces cardiac fibrosis through stimulation of myofibroblast growth, which is mediated by aldosterone and glucose [36, 37].

II. 7. c. vi. Vascular Endothelial Growth Factor (VEGF):

The expression of mRNA and protein for VEGF and its receptors such as VEGF-R1 and VEGF-R2 has been reduced significantly (40%-70%) in a diabetic patients [38]. Because of this the normal molecular process which regulates the angiogenesis has been impaired [39].

II. 7. c. vii. Gene Expression:

Increased expression of genes such as contractile proteins β- Myosin heavy chain (β-MHC), α-skeletal actin and atrial light chain 1 in left ventricular hypertrophy and cardiac failure in some studies [40, 41].

II. 7. c. viii. Endothelial Dysfunction:

Diabetes is associated with anatomical and functional abnormalities of vascular endothelium [42]. Endothelial dysfunction in chronic hyperglycemia contributes to impaired endothelial nitric oxide (NO) production [43], enhanced production of vasoconstrictor prostaglandins, endothelium adhesion molecules, vascular and platelet growth factors and glycated proteins which cumulatively

increases the vasomotor tone and vascular permeability and thereby enhances growth and remodelling.

The other features of endothelial dysfunction includes the weakening of intercellular junctions, protein synthesis alteration, accelerated disappearance of capillary endothelium [44], adhesion glycoproteins production/expression. These alterations on endothelial cells promote the attachment of monocytes and leucocytes. Also transendothelial migration occurs. This chronic hyperglycemia induced endothelial dysfunction also enhances endothelial cell matrix production which further contributes to basement membrane thickening [45]. Endothelial dysfunction can also enhance atherosclerosis in diabetic individuals.

II. 8. ECHOCARDIOGRAPHY AND LV MASS:

Echo sounding was first applied in 1920 by humans for recording of depth in oceanographic studies and submarine detection. By definition "Ultrasound is the sound having a frequency of greater than 20000cycles/second". Ultrasonography device uses a piezoelectric transducer. Use of pulse reflected ultrasound for non destructive testing was first used by firestone.

The use of ultrasonography for examination of heart was first done by Keidal. Dr. Hertz initiated the use of pulse reflected ultrasonography. A number of ultrasonic studies of heart were done by Elder. Feigenbaum H. Popp R.L [46]

and associates (1968) used ultrasonic methods for measuring left ventricular wall thickness.

Measurement of left ventricular wall thickness and mass by echocardiography was first published by Troy, Rackley and Pombo in 1972 [47]. Murray J.A Johnston W, Reid J.W et al showed good results by correlating left ventricular volume and dimension by echocardiography with angiographic studies [48].

Devereux. R (1976) using penn convention method calculated the left ventricular mass by echocardiography and they used angiographic studies [49] for calculation of left ventricular mass.

The echocardiography instrumentation has been complex with evolution from M mode to 2-Dimensional echocardiography. Also from 2D-echocardiography to doppler techniques with colour coding.

A 12-lead electrocardiography is readily available diagnostic modality for left ventricular hypertrophy which is commonly used by many physicians for detection of left ventricular hypertrophy [50]. Sensitivity of electrocardiography for detecting left ventricular hypertrophy is 49% and specificity of 95% by casale's modified electrocardiographic criteria. This limits the use of electrocardiography for detecting left ventricular hypertrophy and hence increases the need for other diagnostic tools [51]. The presence of left

ventricular hypertrophy can be accurately assessed by echocardiography which is difficult to be detected by electrocardiography [52].

Daniel savage et al [53] showed that echocardiography identified left ventricular hypertrophy in 50% of hypertensive patients, whereas electrocardiography identified less than 10% cases. Echocardiography is a reliable and reproducible method for measuring left ventricular mass and left ventricular wall thickness as shown by Bart L. Troy [54]. In hypertensive patients the diagnostic method of choice for measuring left ventricular hypertrophy is echocardiography [55]. M mode echocardiography used for evaluation of left ventricular hypertrophy gives information about both wall thickness and cavity size.

Pearson et al [7] found that M mode echocardiography has sensitivity of 88% and specificity of 84% for detecting left ventricular mass which is better than 2-Dimensional echocardiography.

In Framingham study [56], it is showed that echocardiography has a high sensitivity in predicting risk stratification in left ventricular hypertrophy when compared to electrocardiography. Donna et al (2003) [62] found that for estimating quantitative measurement of left ventricular mass uses echocardiographic chamber dimension and left ventricular wall thickness measurements. While echocardiographic M mode left ventricular images are measured using strip-chart paper tracings and hand-held callipers, in digitized

M-mode left ventricular images direct measurements were made on computer screens using electronic calipers.

II. 9. LEFT VENTRICULAR MASS AND CARDIOVASCULAR MORBIDITY:

Casale et al [57] studied cardiovascular risks of left ventricular hypertrophy. In this study men with left ventricular hypertrophy had a higher risk of cardiovascular morbidity than men without left ventricular hypertrophy of same age, blood pressure and cholesterol levels (24% vs. $p<0.01$).

Koren MJ et al [87] and Verdecchia P et al [88] explained that intermediate risk of target organ damage occurs with eccentric hypertrophy (normal relative wall thickness and increased left ventricular mass) and highest risk of target organ damage occurs with concentric hypertrophy (increased relative wall thickness and left ventricular mass).

There is higher incidence of atherothrombotic brain infarctions and new cardiac events in patients with echocardiographic evidence of left ventricular hypertrophy. There occurs more frequent episodes of complex ventricular ectopics in patients with left ventricular hypertrophy than without left ventricular hypertrophy. Levy et al [56,58,59] in their Framingham study of 6218 patients showed that complex ventricular arrhythmias occurs in patients with echocardiographic evidence of left ventricular hypertrophy. There is a six-fold

increase in coronary mortality and eight-fold increase in cardiovascular mortality in patients with left ventricular hypertrophy [56]

Adewole et al (2006) [64] showed that left ventricular hypertrophy is an independent risk factor for cardiovascular events. It is shown that combination of relative wall thickness and left ventricular mass identified different forms of left ventricular geometry. The prognosis is worst with concentric hypertrophy.

Paolo verdecchia et al (2001) [67] showed that in uncomplicated patients with essential hypertension, echocardiographic demonstration of left ventricular mass as a prognostic value and they found a strong relation between left ventricular mass and cardiovascular morbidity.

Systolic and diastolic function abnormalities occur due to increased left ventricular mass [69, 70, 71]. The thickness of left ventricle shown by 2-Dimensional echocardiography correlates with left ventricular mass. Normal thickness ranges from 0.6 to 1.1 centimetres and when the thickness is >1.1cm there is associated left ventricular hypertrophy.

High blood pressure may account for 37% of total cardiovascular diseases in men and 27% in women, 30% of myocardial infarction in women and 14% in men, 59% of chronic heart failure in women and 39% in men, 35% of ischemic strokes and 56% of chronic kidney diseases.

Randomised controlled trials have proved that reducing blood pressure with antihypertensive treatment reduces total mortality of coronary heart diseases, heart failure, stroke and chronic kidney diseases.

Okin et al (2007) [68] showed that with antihypertensive therapy there is regression of electrocardiographic left ventricular hypertrophy and it is associated with decreased incidence of atrial fibrillation.

III. AIM OF THE STUDY

- To compare the left ventricular mass in hypertensive patients with diabetes mellitus and without diabetes mellitus.
- To compare these two groups with controls.

IV. BACKGROUND

IV. 1. Selection of subjects:

Patients with hypertension alone and patients with both hypertension and diabetes mellitus of more than 35 years attending the hypertension outpatient department of kilpauk medical college hospital. The selected patients should be on regular treatment not on cardiac remodelling drugs like angiotensin converting enzyme inhibitors and aldosterone antagonists. Control groups with more than 35 years without diabetes mellitus and hypertension.

IV. 2. Inclusion Criteria:

- All patients > 35 yrs of age with hypertension of duration > 5yrs on regular treatment.
- Patients >35yrs of age with hypertension and diabetes mellitus >5yrs duration on regular treatment.
- Healthy controls of >35yrs of age.

IV. 3. EXCLUSION CRITERIA:

- Coronary Artery Disease
- Valvular Heart Disease
- Chronic Kidney Disease of non diabetic origin
- Obesity
- Cardiomyopathies

V. MATERIALS AND METHODS:

Setting: Kilpauk Medical College.

Study design: Prospective Cross sectional study

Period of study: 6 months from March 2013 to August 2013.

Sample size: 150 subjects (50 Hypertensive cases + 50 Cases of both hypertension and diabetes + 50 controls).

V. 1. Both cases and controls are investigated by following measures:

- Proper history and past medical history and demographic details were collected.
- General examination , Vitals monitoring includes blood pressure and pulse rate
- Body Mass Index
- Fasting and Post Prandial Blood Sugar
- Complete hemogram
- Blood Urea, Serum Creatinine and Serum Electrolytes Urine analysis
- Serum Total Cholesterol, Serum Triglyceride levels.
- Electrocardiography
- Chest X-Ray
- 2-Dimensional Echocardiography

V. 2. Echocardiography:

By using transthoracic 2-Dimensional echocardiographic method,

the following left ventricular dimensions are measured by M-mode technique using the parasternal long axis view just above/at the tip of the papillary muscle level.

- Left ventricular internal diameter at end diastole (LVID-D)
- Interventricular septal thickness at end diastole (IVST-D)
- Posterior wall thickness at end diastole (PWT-D)

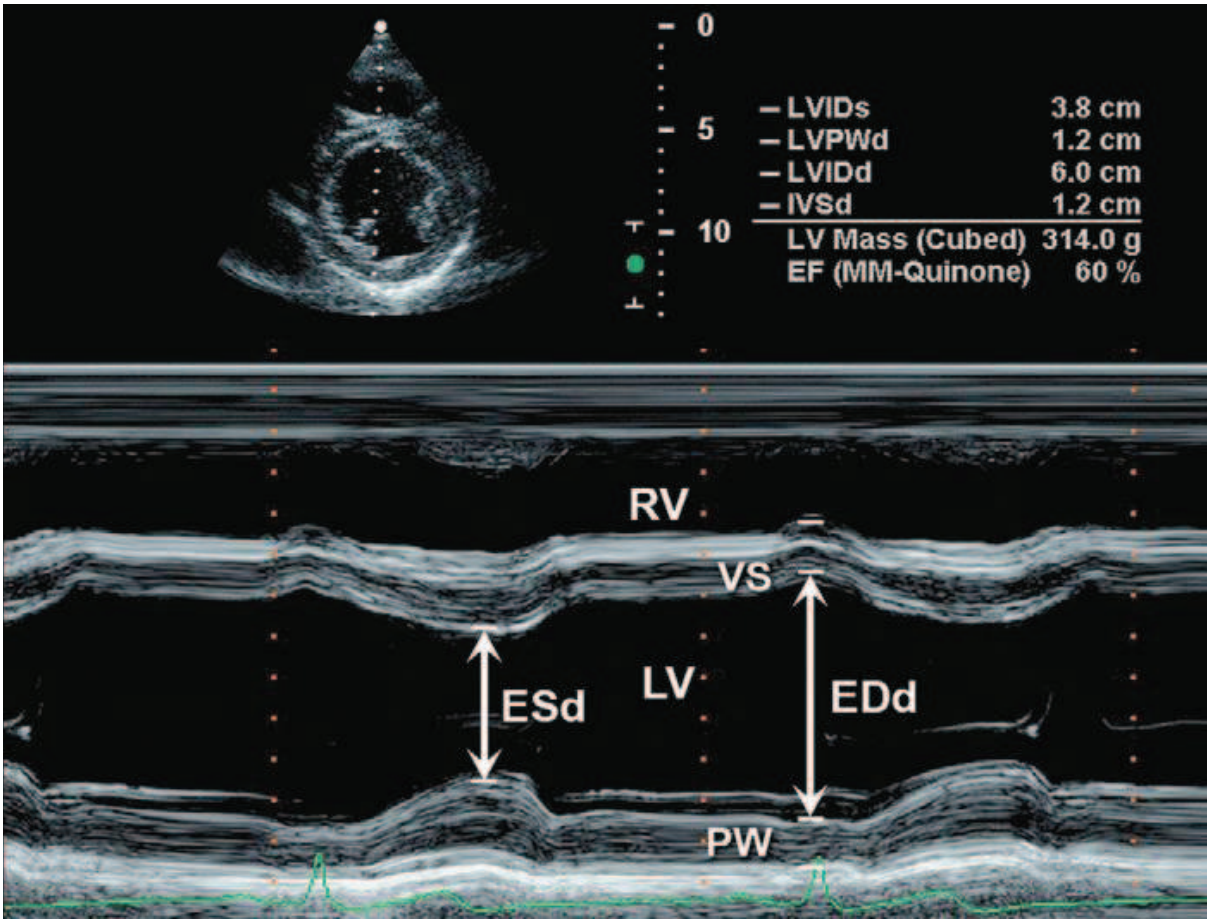


FIGURE 10: Two dimensional guided M mode echocardiogram of left ventricle at papillary muscle level [73]

Apart from this measurements, left systolic functions, diastolic functions and ejection fractions also measured.

Left ventricular mass was calculated by penn convention method which was first used by Devereux and colleagues during 1981. They found that this formula best correlates with anatomic left ventricular mass.

LV mass = $1.04[(\text{LVID-D+IVST-D+PWT-D})^3 - (\text{LVID-D})^3]$-13.6gm.

	Women				Men			
		ABNORMAL				ABNORMAL		
MEASURE	REFERENCE RANGE	MILDLY	MODERATELY	SEVERELY	REFERENCE RANGE	MILDLY	MODERATELY	SEVERELY
Linear method								
LV mass, g	67-162	163-186	187-210	≥211	88-224	225-258	259-292	≥293
LV mass/BSA, g/m²	**43-95**	**96-108**	**109-121**	**≥122**	**49-115**	**116-131**	**132-148**	**≥149**
LV mass/height, g/m	41-99	100-115	116-128	≥129	52-126	127-144	145-162	≥163
LV mass/height, g/m	18-44	45-51	52-58	≥59	20-48	49-55	56-63	≥64
Relative wall thickness, cm	0.22-0.42	0.43-0.47	0.48-0.52	≥0.53	0.24-0.42	0.43-0.46	0.47-0.51	≥0.52
Septal thickness, cm	**0.6-0.9**	**1.0-1.2**	**1.3-1.5**	**≥1.6**	**0.6-1.0**	**1.1-1.3**	**1.4-1.6**	**≥1.7**
Posterior wall thickness, cm	**0.6-0.9**	**1.0-1.2**	**1.3-1.5**	**≥1.6**	**0.6-1.0**	**1.1-1.3**	**1.4-1.6**	**≥1.7**
Two-dimensional method								
LV mass, g	66-150	151-171	172-182	≥183	96-200	201-227	228-254	≥255
LV mass/BSA, g/m²	**44-88**	**89-100**	**101-112**	**≥113**	**50-102**	**103-116**	**117-130**	**≥131**

TABLE 3: Reference values of left ventricular mass [72]:

V. 3. Statistical Analysis:

Mean values of all parameters in groups were calculated by independent sample t-test. To compare the distributions of dichotomous data viz., age, gender, body mass index, smoking, alcoholism, duration of hypertension and diabetes mellitus and left ventricular mass, Chi-square test was used. ANOVA test was used for comparing mean LV mass in between three groups. Association of LV mass between hypertension group and group of both hypertension and diabetes mellitus was assessed by logistic regression model.

All statistical analysis were performed using SPSS (Software Package used for Statistical Analysis) package. A p- value of less than 0.05 was considered to be statistically significant.

VI. OBSERVATION ANALYSIS:

Table 1: Age wise distribution of cases (hypertensive groups and groups of both hypertension and diabetes) and controls:

			Group		
			Control	HT	HT + DM
Age in years	36-45	Count	13	8	7
		% within Group	26.0%	16.0%	14.0%
	46-55	Count	28	16	12
		% within Group	56.0%	32.0%	24.0%
	Above 55	Count	9	26	31
		% within Group	18.0%	52.0%	62.0%

The minimum age of the cases and controls was 36 years and maximum age was 85 years. Among 50 hypertensive cases 16% were in 36-45yrs, 32% were in 46-55yrs, 52% were in above 55yrs with mean age of 58.06 yrs. Likewise among 50 cases with both hypertension and diabetes mellitus, there were 14%, 24% and 62% with mean age of 58.44yrs and among 50 controls there were 26%, 56% and 18% with mean age of 49 yrs in each of the above age groups respectively.

Chart 1: Age wise distribution of cases (hypertensive groups and groups of both hypertension and diabetes) and controls:

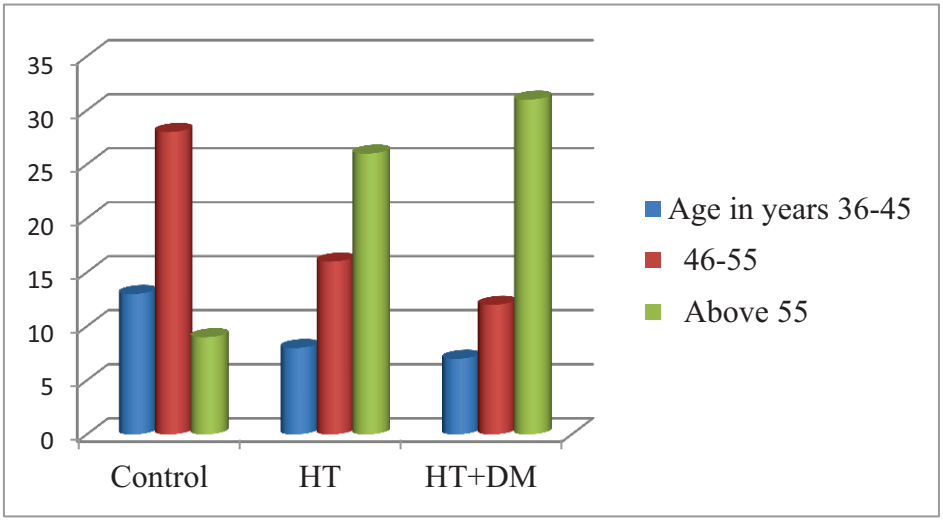


Table: 2: Age wise distribution of left ventricular mass in three groups:

Group			Age in years			p value
			36-45	46-55	Above 55	
Control	LV mass	Normal	13	27	8	0.248
		Mild	0	0	1	
		Moderate	0	1	0	
HT	LV mass	Normal	5	9	9	0.543
		Mild	0	3	6	
		Moderate	0	1	2	
		Severe	3	3	9	
HT +DM	LV mass	Normal	5	4	4	0.039
		Mild	0	2	5	
		Moderate	0	2	2	
		Severe	2	4	20	

From the above table (Table:2), there is significant statistical correlation between left ventricular mass and age group in cases with both hypertension and diabetes group with the p value of 0.039.The left ventricular mass increases with age in this group. But in control group and cases with hypertension only group, the p value was 0.248 and 0.543 respectively and it was not statistically significant.

Chart 2: Age wise distribution of left ventricular mass in three groups:

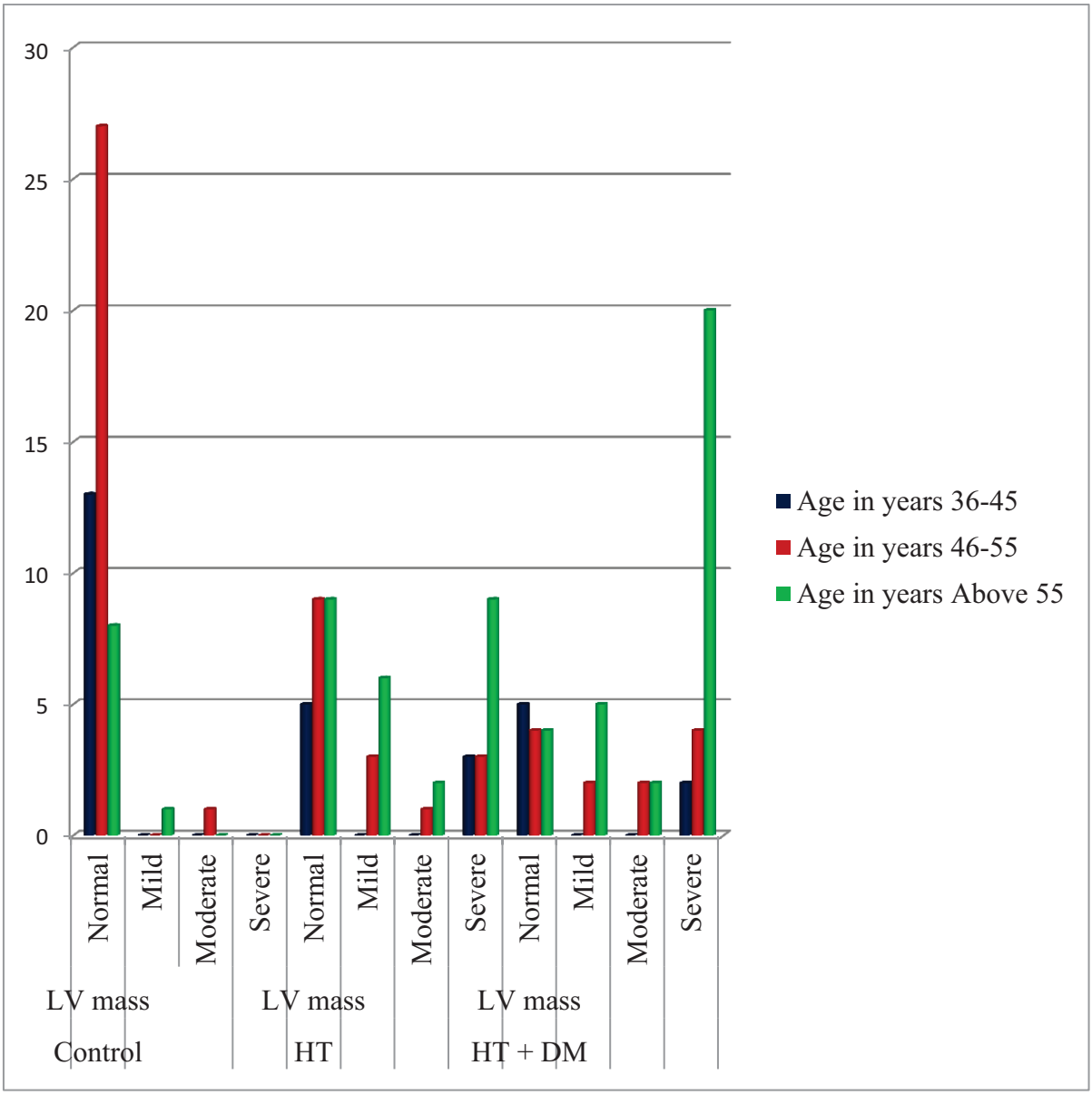


Table 3: Sex wise distribution of cases in of cases (Hypertensive groups and groups of both Hypertension and Diabetes) and controls:

			Group		
			Control	HT	HT + DM
Sex	Male	Count	21	23	22
		% within Group	42.0%	46.0%	44.0%
	Female	Count	29	27	28
		% within Group	58.0%	54.0%	56.0%

Among 50 cases with hypertension, 23 were male and 27 were female, i.e., 46% and 54% respectively. Likewise, among 50 cases with both hypertension and diabetes mellitus 22(44%) were male and 28(56%) were females. Among controls 21(42%) were male and 29(58%) were females.

Chart 3: Sex wise distribution of cases in three groups:

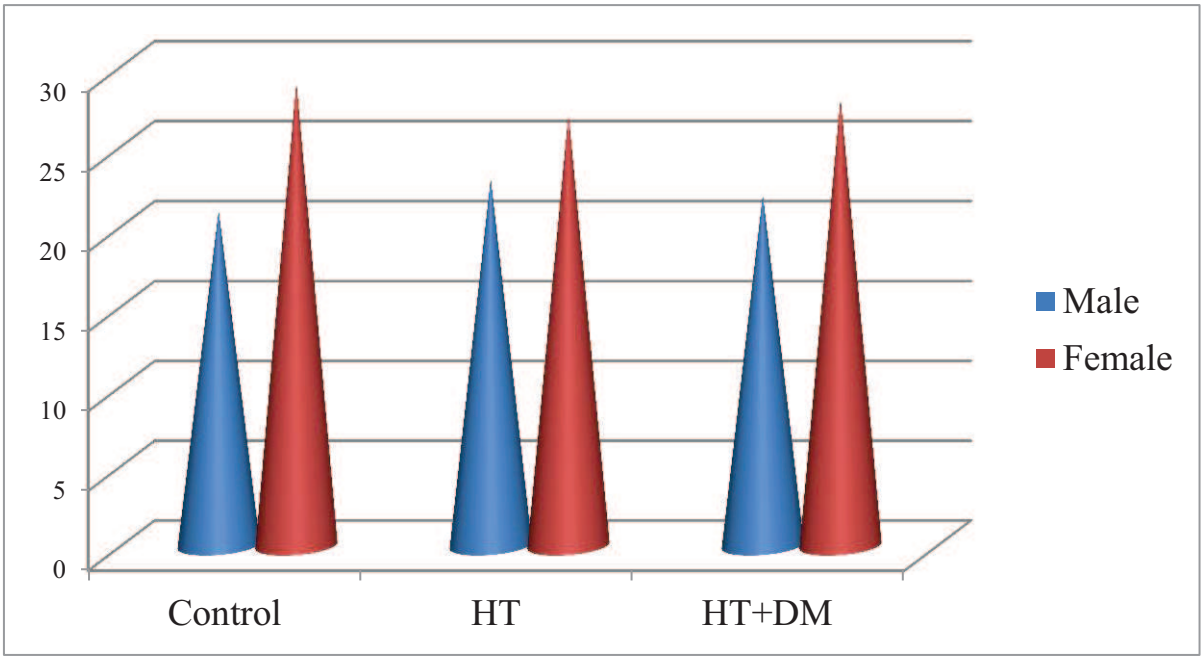


Table 4: Sex wise distribution of left ventricular mass in three groups:

Sex			Group			p value
			Control	HT	HT+DM	
Male	LV mass	Normal	20	12	8	0.286
		Mild	1	4	4	
		Moderate	0	0	3	
		Severe	0	7	7	
Female	LV mass	Normal	28	11	5	0.042
		Mild	0	5	3	
		Moderate	1	3	1	
		Severe	0	8	19	

Among 23 male cases with hypertension normal left ventricular mass were 12(52.2%), mildly abnormal were 4(17.4%), moderately abnormal were 0(0%), severely abnormal were 7(30.4%). Likewise among 22 male cases with hypertension and diabetes, normal were 8(36.4%), mild, moderate and severely abnormal were 4(18.2%), 3(13.6%), 7(31.8%) respectively. Among 21 male controls 20(95.2%) were normal, 1(4.8%) were mildly abnormal.

Among 27 female cases with hypertension normal were 11(40.7%), mild, moderate and severely abnormal were 5(18.5%), 3(11.1%), 8(29.6%) respectively. Among 28 female cases with hypertension and diabetes 5(17.9%) were normal, mild, moderate and severely abnormal were 3(10.7%), 1(3.6%),

19(67.9%) respectively. Among 29 female controls 28 were normal and 1 was moderately abnormal.

From the above table (Table: 4), female gender has significant correlation with severity of LV mass (p = 0.042). The following bar diagram (Chart: 4) explains that there is increased LV mass in females with both hypertension and diabetes groups when compared to other two groups. But there is no significant statistical correlation between male gender and left ventricular mass (p = 0.286).

Chart 4: Sex wise distribution of left ventricular mass in three groups:

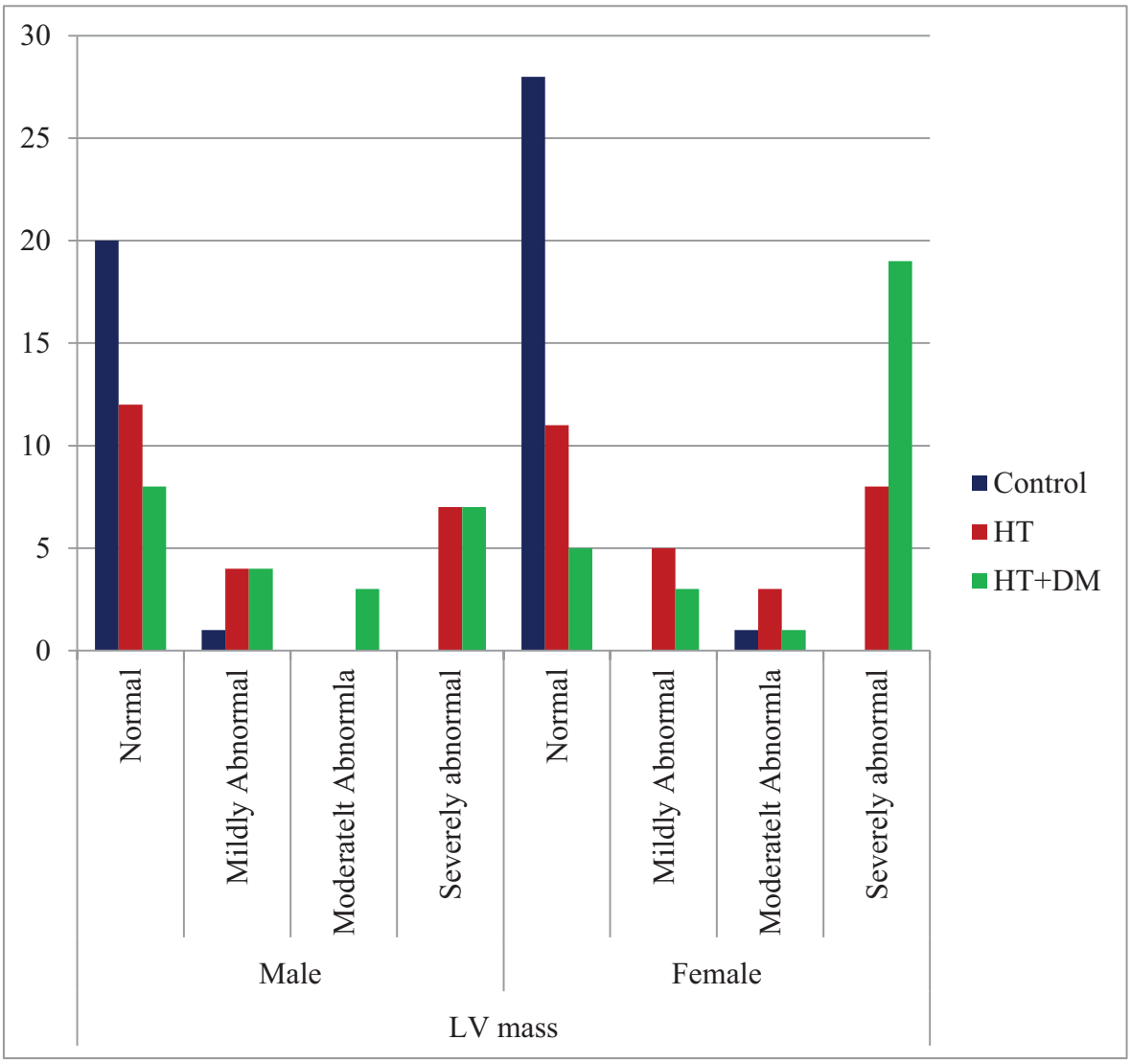


Table 5: BMI wise distribution of cases and controls in three groups:

			Group		
			Control	HT	HT + DM
BMI	Normal	Count	31	27	24
		% within Group	62.0%	54.0%	48.0%
	Overweight	Count	19	23	26
		% within Group	38.0%	46.0%	52.0%

Among 50 cases with hypertension 27 were having normal BMI, 23 were having overweight. Likewise cases with both hypertension and diabetes 24 were normal BMI, 26 were having overweight. Among 50 controls 31 were normal BMI, 19 were overweight.

Chart 5: BMI wise distribution of cases and controls:

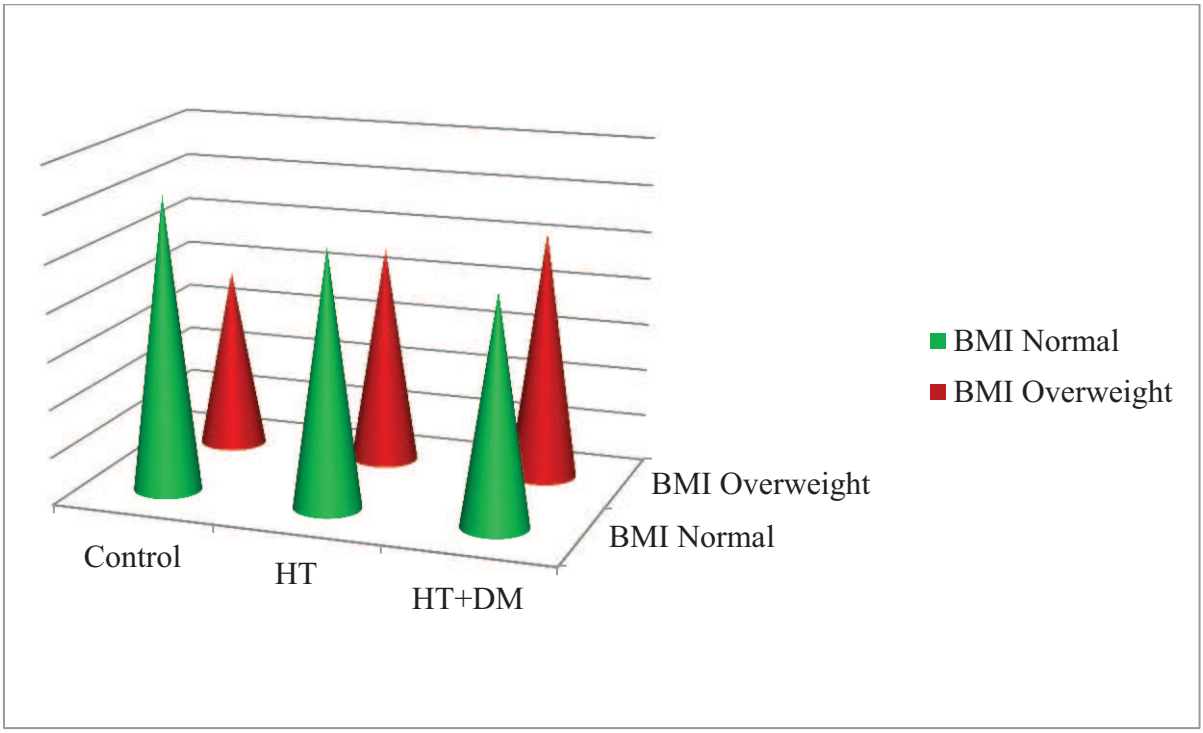


Table 6: BMI wise distribution of left ventricular mass in three groups:

Group			BMI		
			Normal	Overweight	p value
Control	LV mass	Normal	30	18	0.325
		Mild	1	0	
		Moderate	0	1	
		Severe	0	0	
HT	LV mass	Normal	14	9	0.084
		Mild	2	7	
		Moderate	3	0	
		Severe	8	7	
HT+ DM	LV mass	Normal	7	6	0.624
		Mild	3	4	
		Moderate	3	1	
		Severe	11	15	

Among cases with hypertension with abnormal BMI, 7(30.4%) had severely abnormal left ventricular mass. In the same way cases with hypertension and diabetes with abnormal BMI, 15(57.7%) had severely abnormal left ventricular mass. Likewise controls, only 0% have abnormal or severely left ventricular mass.

From the above table (Table: 6) compared to hypertensive cases, cases with hypertension and diabetes group have more percentage of cases having severe left ventricular mass increases with increase in BMI, but this is not significant statistically ($p > 0.05$).

Chart: 6: BMI wise distribution of LV mass in three groups:

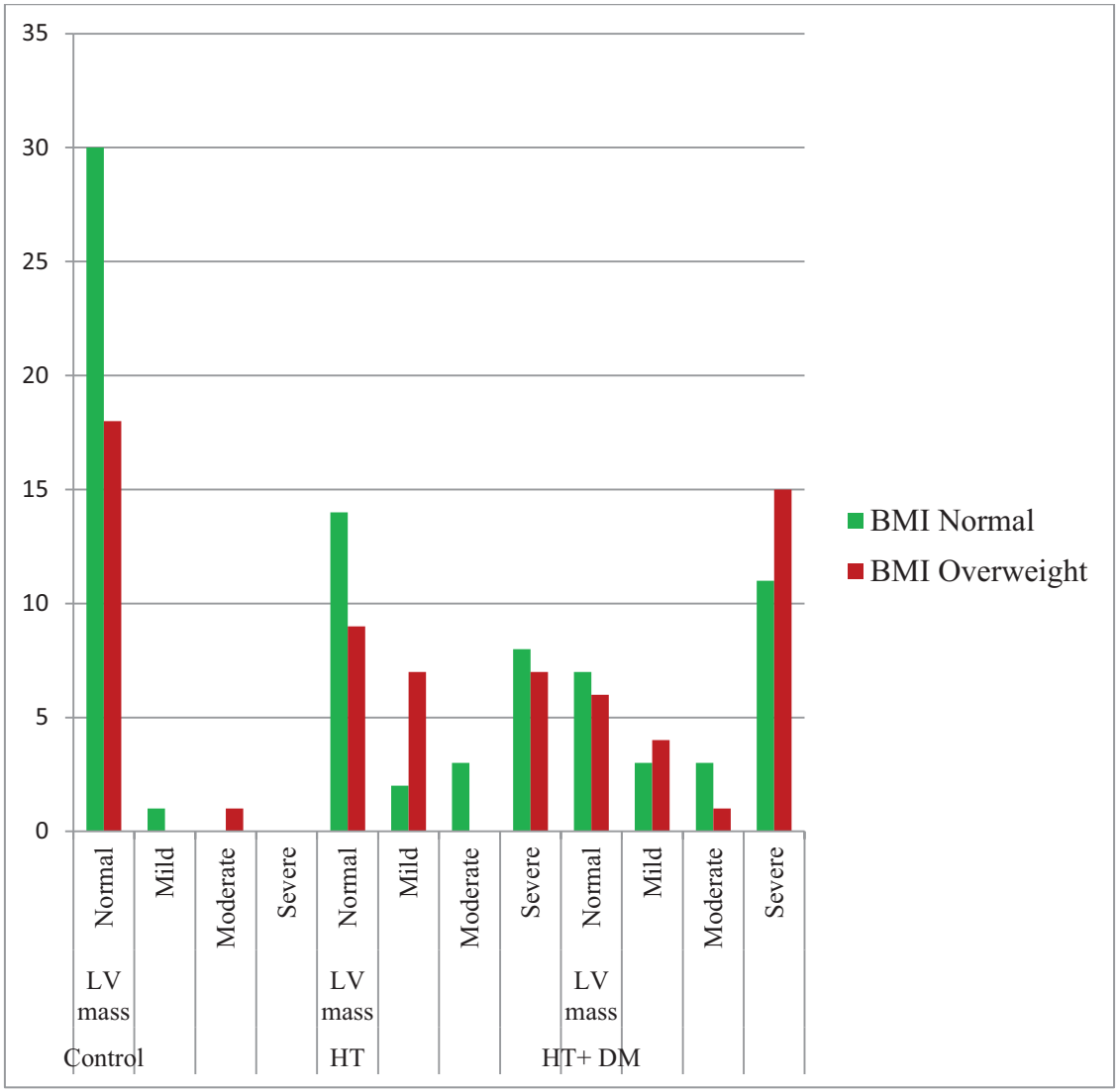


Table 7: Distribution of cases according to the Duration of HT/HT+DM:

			Group	
			HT	HT + DM
Duration of HT	6-10	Count	40	33
		% within Group	80.0%	66.0%
	11-15	Count	6	11
		% within Group	12.0%	22.0%
	Above 15	Count	4	6
		% within Group	8.0%	12.0%

Among 50 cases with hypertension, 4 (8%) were have more than 15 years duration of hypertension. Likewise cases with both hypertension and diabetes 6(12%) have more than 15 years duration of hypertension and diabetes.

Chart 7: Distribution of cases according to the duration of HT/HT+DM:

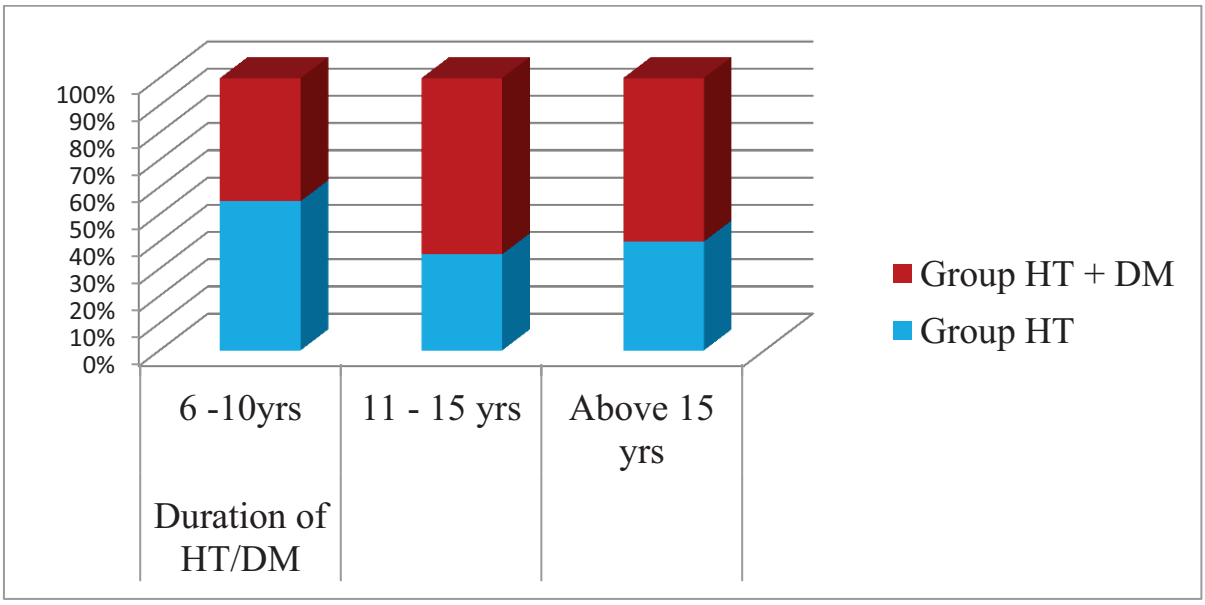


Table 8: Distribution of LV mass according to the duration of HT/HT+DM:

Group			Duration of HT/DM			p value
			6-10yrs	11-15yrs	Above 15yrs	
HT	LV mass	Normal	17	2	4	0.378
		Mild	8	1	0	
		Moderate	2	1	0	
		Severe	13	2	0	
HT + DM	LV mass	Normal	9	3	1	0.446
		Mild	4	1	2	
		Moderate	1	2	1	
		Severe	19	5	2	

Among 50 cases with hypertension 4(8%) have >15yrs duration of hypertension and all these cases had normal LV mass. Likewise among cases with hypertension and diabetes 6(12%) have >15yrs duration, within this 2 (33.3%) have severely abnormal left ventricular mass. There is no significant statistical correlation between duration of HT/DM and LV mass in two groups ($p>0.05$).

Chart 8: Distribution of LV mass according to the duration of HT/HT+DM:

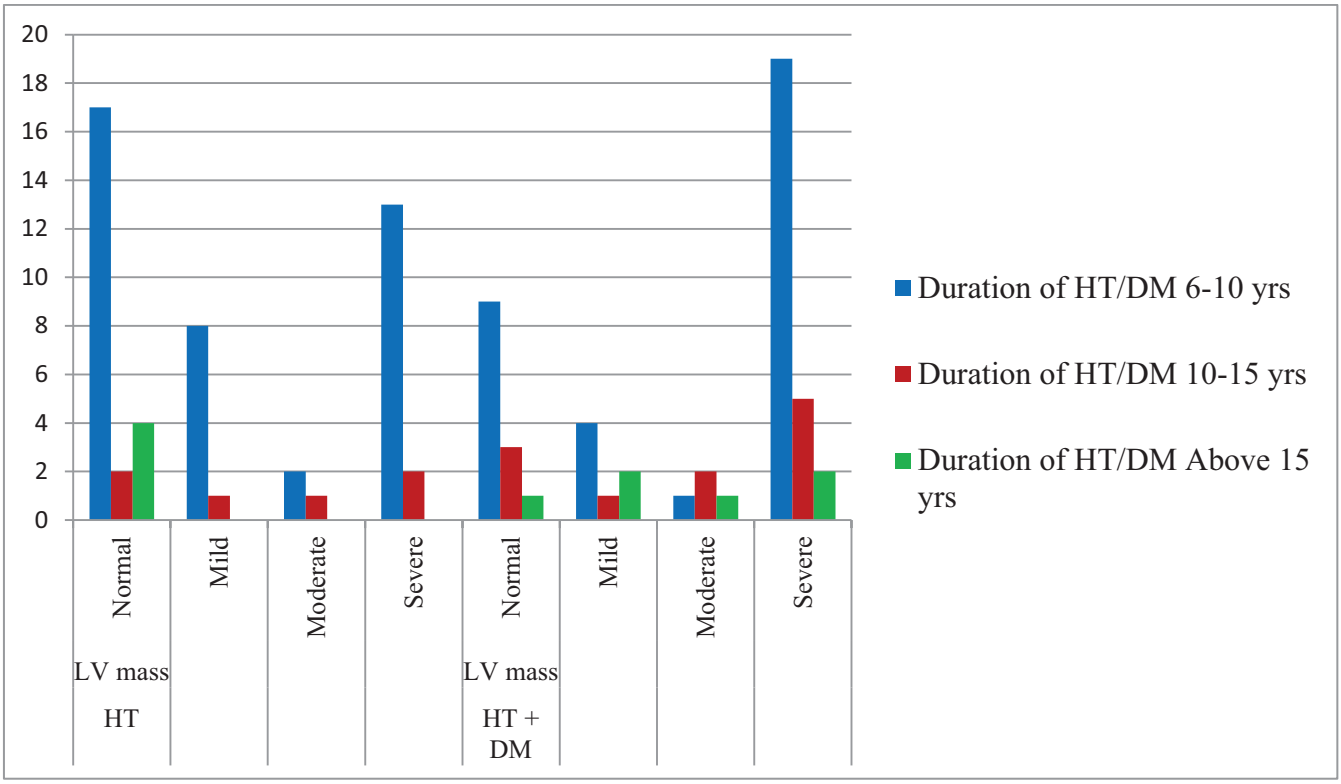


Table: 9: Mean LV mass according to the duration of HT in HT only group:

		LV mass			
		Mean	SD	Minimum	Maximum
Duration of HT	6-10	202.00	64.06	88.47	379.37
	11-15	203.99	66.07	102.48	319.17
	Above 15	192.83	72.17	110.91	330.65

The mean LV mass in hypertension only group was nearly same in various durations of hypertension.

Table: 10: Mean LV mass according to the duration of HT/DM in Both HT and DM group:

		LV mass			
		Mean	SD	Minimum	Maximum
Duration of HT/DM	6-10	214.52	66.05	88.47	379.37
	11-15	204.29	62.82	102.48	296.70
	Above 15	229.42	68.84	152.47	330.65

The mean LV mass was increasing from 214.52gm to 229.42gm with increase in the duration of HT and DM in the group with both hypertension and diabetes.

Table 11: Distribution of DD among three groups:

			Group		
			Control	HT	HT + DM
DD	No DD	Count	44	20	16
		% within Group	88%	40.0%	32.0%
	Grade1DD	Count	6	30	34
		% within Group	12%	60.0%	68.0%

Among 50 hypertensives 30(60%) had grade 1DD, likewise 34(68%) had grade 1 DD in cases with both hypertension and diabetes. Among controls 6(12%) having grade 1 DD. There is significant statistical correlation (p=0.001)

between DD and cases with hypertension with diabetes group compared to others.

Chart 9: Distribution of DD among three groups:

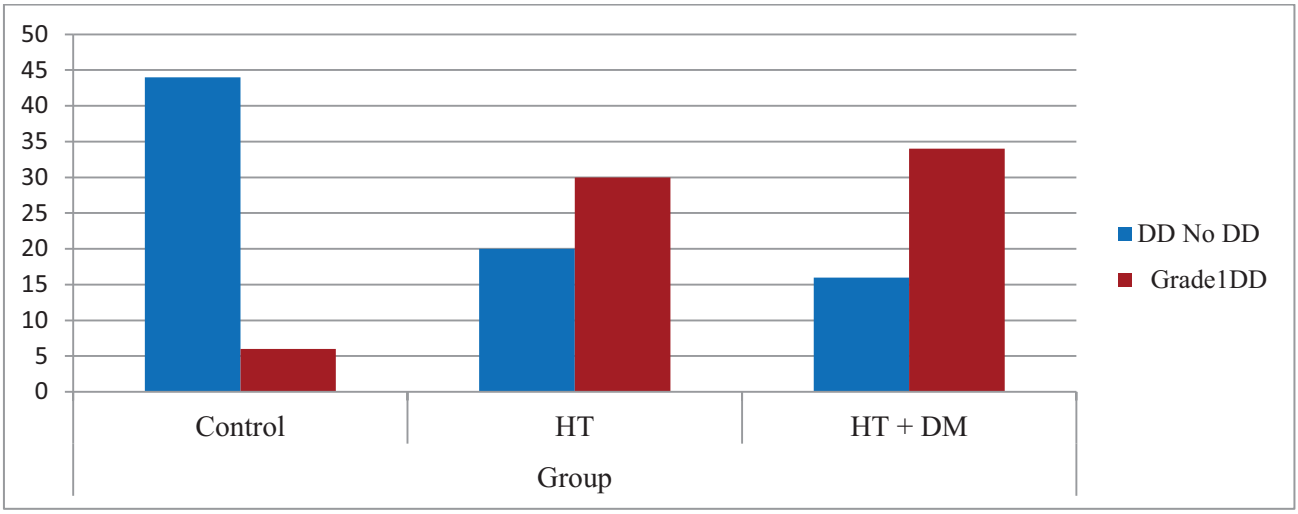


Table 12: Correlation DD and LV mass among three groups:

Group			DD		p value
			No DD	Grade 1 DD	
Control	LV mass	Normal	44	4	0.001
		Mild	0	1	
		Moderate	0	1	
		severe	0	0	
HT	LV mass	Normal	9	14	0.128
		Mild	2	7	
		Moderate	3	0	
		Severe	6	9	
HT + DM	LV mass	Normal	4	9	0.731
		Mild	3	4	
		Moderate	2	2	
		Severe	7	19	

From the above table (Table:12) 15 cases with hypertension and diabetes group had severely abnormal left ventricular mass 9 cases had grade 1 DD, likewise cases with both hypertension and diabetes group, 26 of severely abnormal left ventricular mass 19 have grade 1DD. So there is significantly increased number of cases with hypertension and diabetes group with increased left ventricular mass have diastolic dysfunction. But there is no statistical correlation between DD and LV mass in groups (HT, HT+DM) with p value of > 0.05.

Chart 10: Correlation DD and LV mass among three groups:

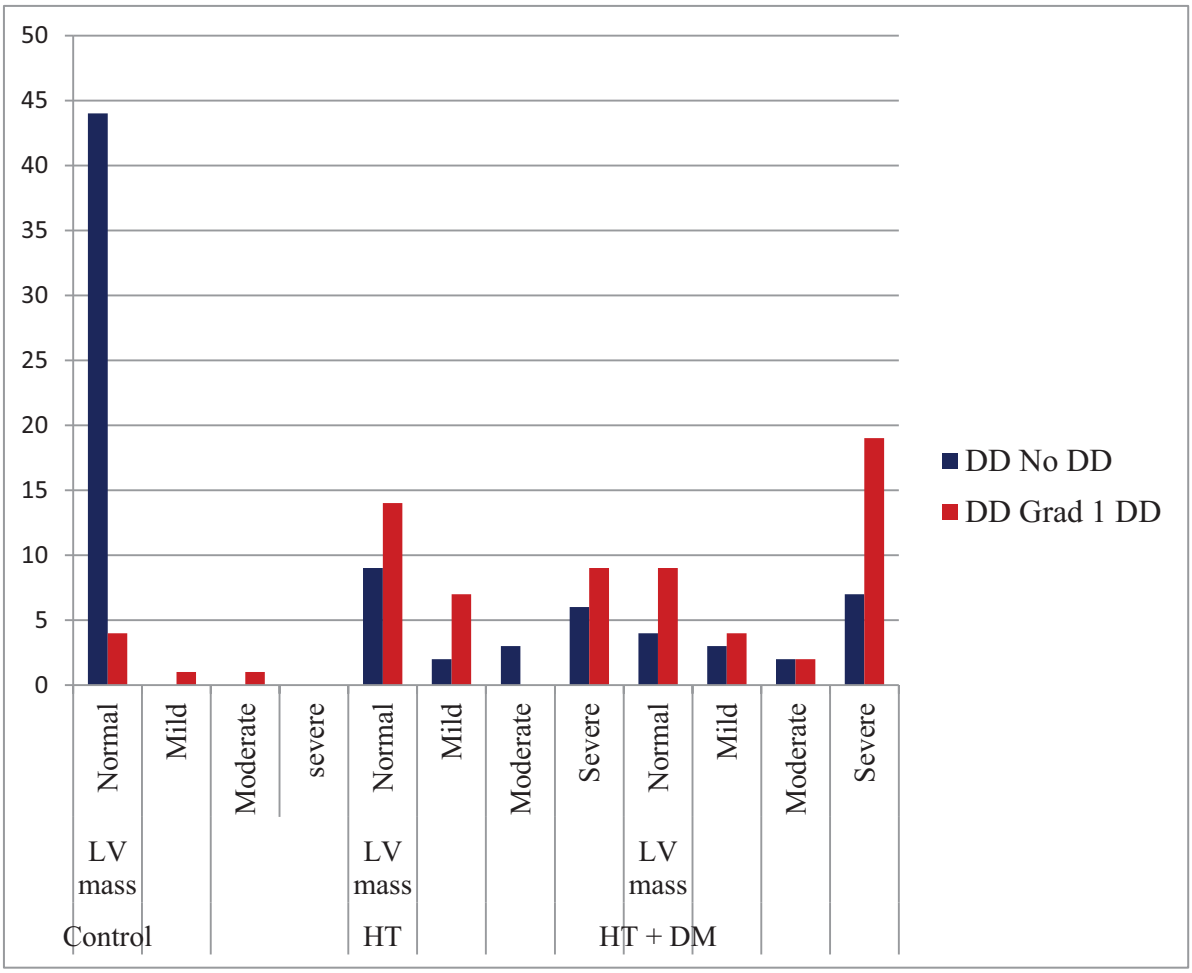


Table 13: Distribution of smoking among three groups:

			Group		
			Control	HT	HT + DM
Smoking	Yes	Count	6	9	6
		% within Group	12.0%	18.0%	12.0%
	No	Count	44	41	44
		% within Group	88.0%	82.0%	88.0%

Among hypertensive groups 9(18%), likewise in groups of both hypertension and diabetes 6(12%), and in controls 6(12%) were having history of smoking.

Chart 11: Distribution of smoking among three groups:

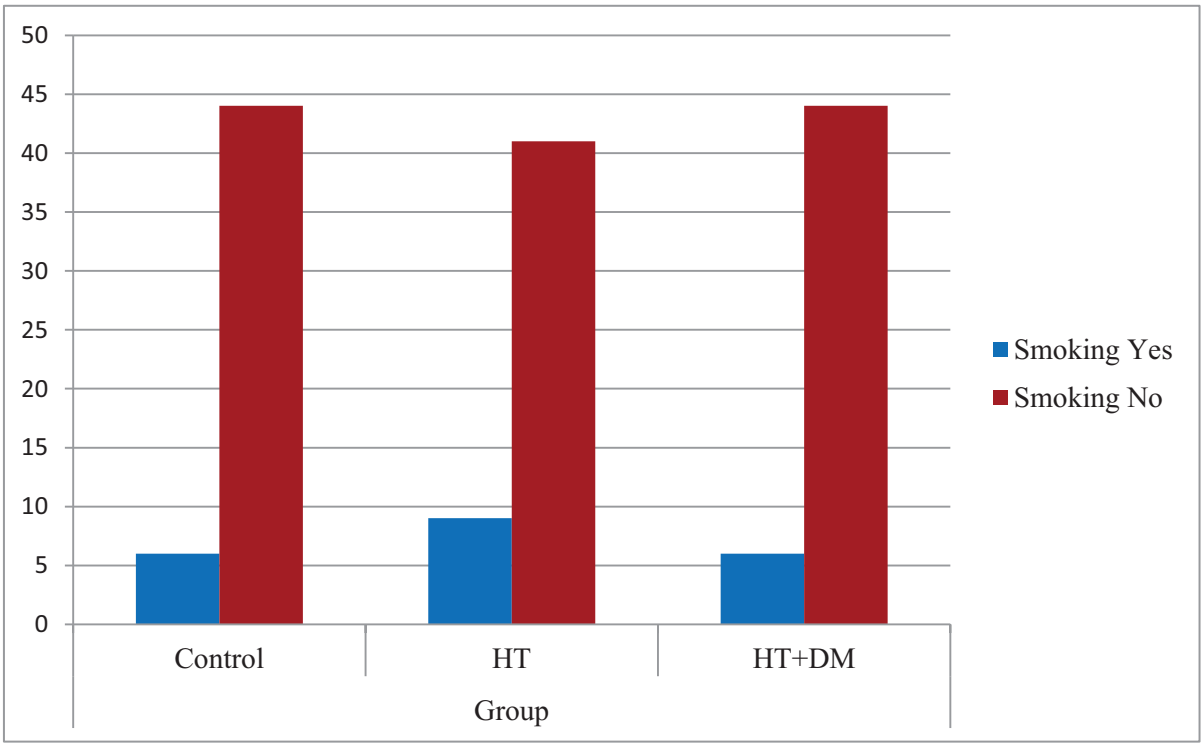


Table 14: Correlation of smoking and LV mass among three groups:

				Control	HT	HT+DM
Smoking	Yes	LV mass	Normal	6	6	3
			Mild	0	2	0
			Moderate	0	0	1
			Severe	0	1	2
	No	LV mass	Normal	42	17	10
			Mild	1	7	7
			Moderate	1	3	3
			Severe	0	14	24

Among 9 smokers in hypertensive groups, 1 have severely abnormal LV mass, likewise among 6 smokers of both hypertensive and diabetics group 2 have severely abnormal LV mass. There is no statistical correlation ($p>0.05$) between smoking and LV mass.

Chart 12: Correlation of smoking and LV mass among three groups:

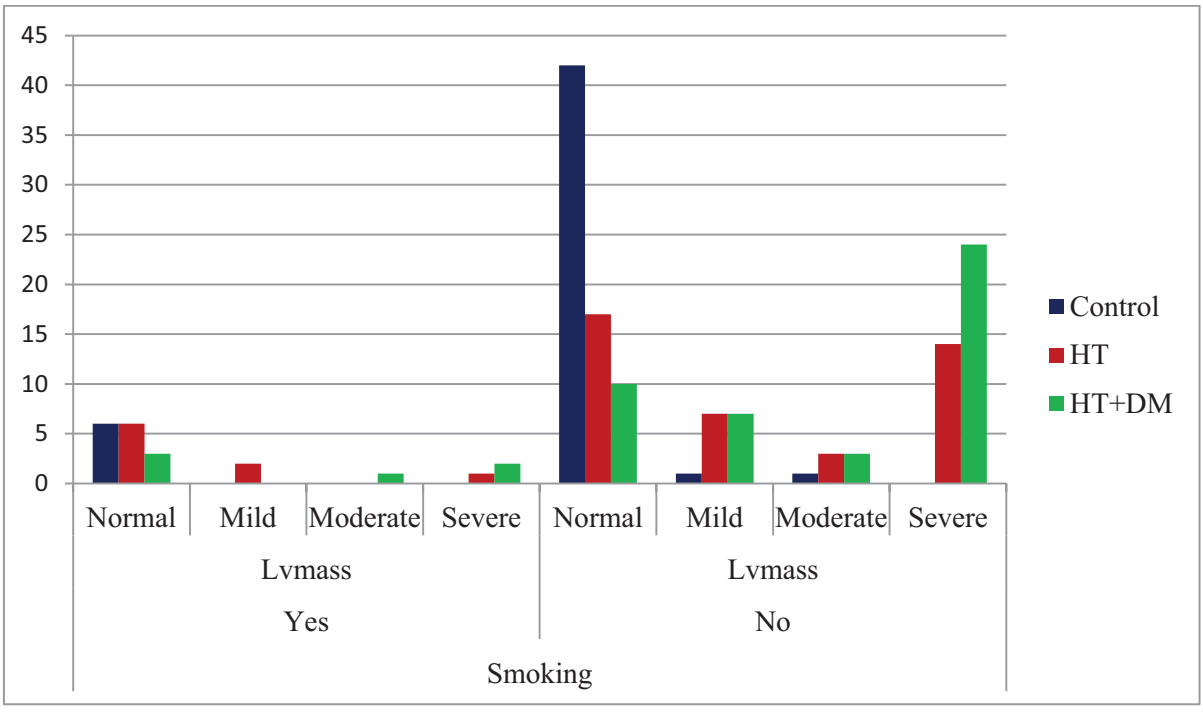


Table 15: Distribution of Alcoholism among three groups:

			Group		
			Control	HT	HT + DM
Alcoholism	Yes	Count	6	9	7
		% within Group	12%	18.0%	14.0%
	No	Count	44	41	43
		% within Group	88%	82.0%	86.0%

Among the 22 of alcoholics, 9(40.9%) were in hypertensive group, 7 (31.8%) were in both hypertensive and diabetics group and 6 were in control group.

Chart 13: Distribution of Alcoholism among three groups:

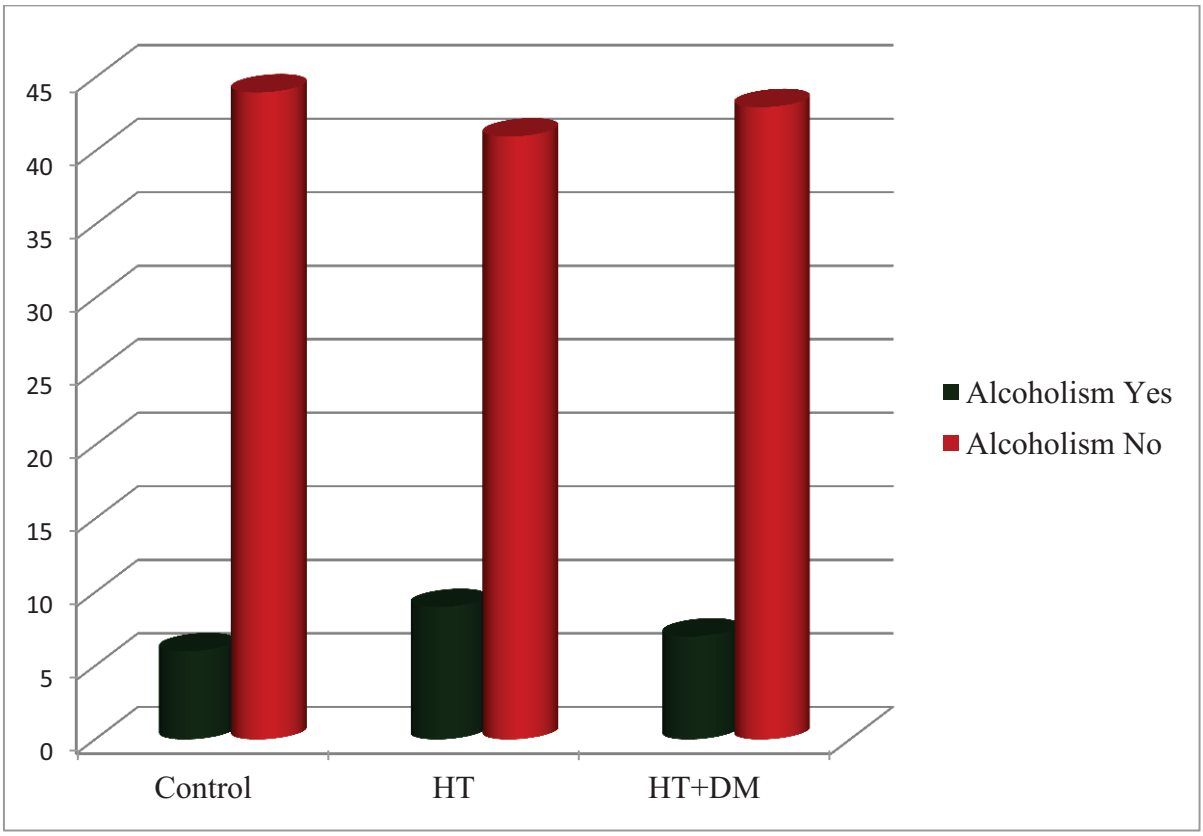


Table 16: Correlation of Alcoholism and LV mass among three groups:

				Control	HT	HT+DM
Alcoholism	Yes	LV mass	Normal	6	7	6
			Mild	0	1	0
			Moderate	0	0	1
			Severe	0	1	0
	No	LV mass	Normal	42	16	7
			Mild	1	8	7
			Moderate	1	3	3
			Severe	0	14	26

Among 9 alcoholics of hypertensive cases 1 have severely abnormal LV mass, likewise among 7 alcoholics of both hypertensive diabetics group only 1 have moderately abnormal LV mass, none have severely abnormal LV mass. There is no statistical significance ($p>0.05$) between LV mass and alcoholism.

Chart 14: Correlation of Alcoholism and LV mass among three groups:

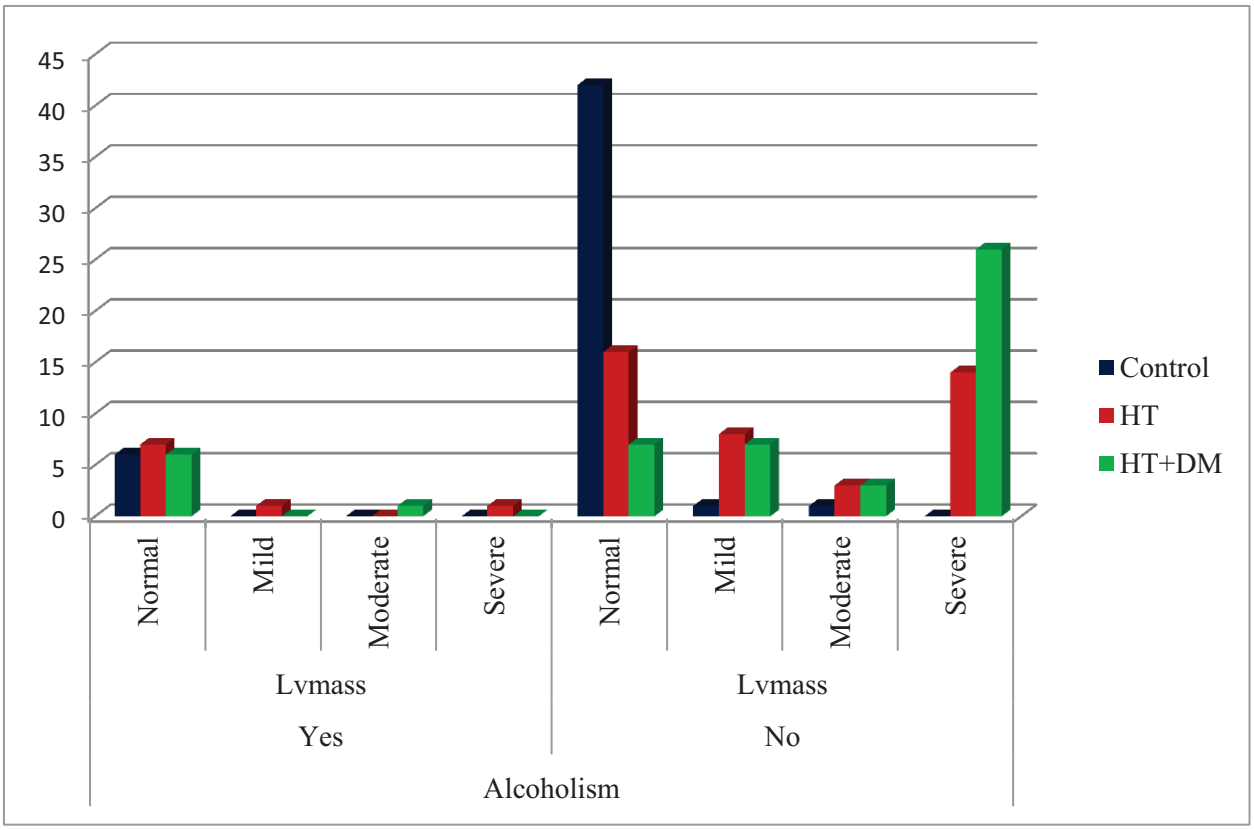


Table 17: Comparison of LV mass between three groups (HT, HT+DM):

			Group			
			Controls	HT	HT+DM	Total
LV mass						
	Normal	Count	48	23	13	84
		% within LV mass	57.1%	27.4%	15.5%	100.0%
		% within Group	96.0%	46.0%	26.0%	56.0%
	Mild	Count	1	9	7	17
		% within LV mass	5.9%	52.9%	41.2%	100.0%
		% within Group	2.0%	18.0%	14.0%	11.3%
	Moderate	Count	1	3	4	8
		% within LV mass	12.5%	37.5%	50.0%	100.0%
		% within Group	2.0%	6.0%	8.0%	5.3%
	Severe	Count	0	15	26	41
		% within LV mass	.0%	36.6%	63.4%	100.0%
		% within group .0%	.0%	30%	52%	27.3%
Total		Count	50	50	50	150
		% within LV mass	33.3%	33.3%	33.3%	100.0%
		% within Group	100.0%	100.0%	100.0%	100.0%

Among hypertensive groups 27(54%) have abnormally increased LV mass, likewise in hypertensive diabetic groups 37(74%) have abnormal LV mass compared to 2(4%) in control groups.

Table 18: Statistical comparison (Chi- Square Test) of LV mass in three groups:

	Value	Df	p value
Pearson Chi-Square	56.009(a)	6	.000
Likelihood Ratio	68.787	6	.000
Linear-by-Linear Association	47.189	1	.000
N of Valid Cases	150		

p value is 0.001, so there is significant statistical correlation of LV mass between these three groups.

Chart 15: Comparison of LV mass between three groups (HT, HT+DM, Controls):

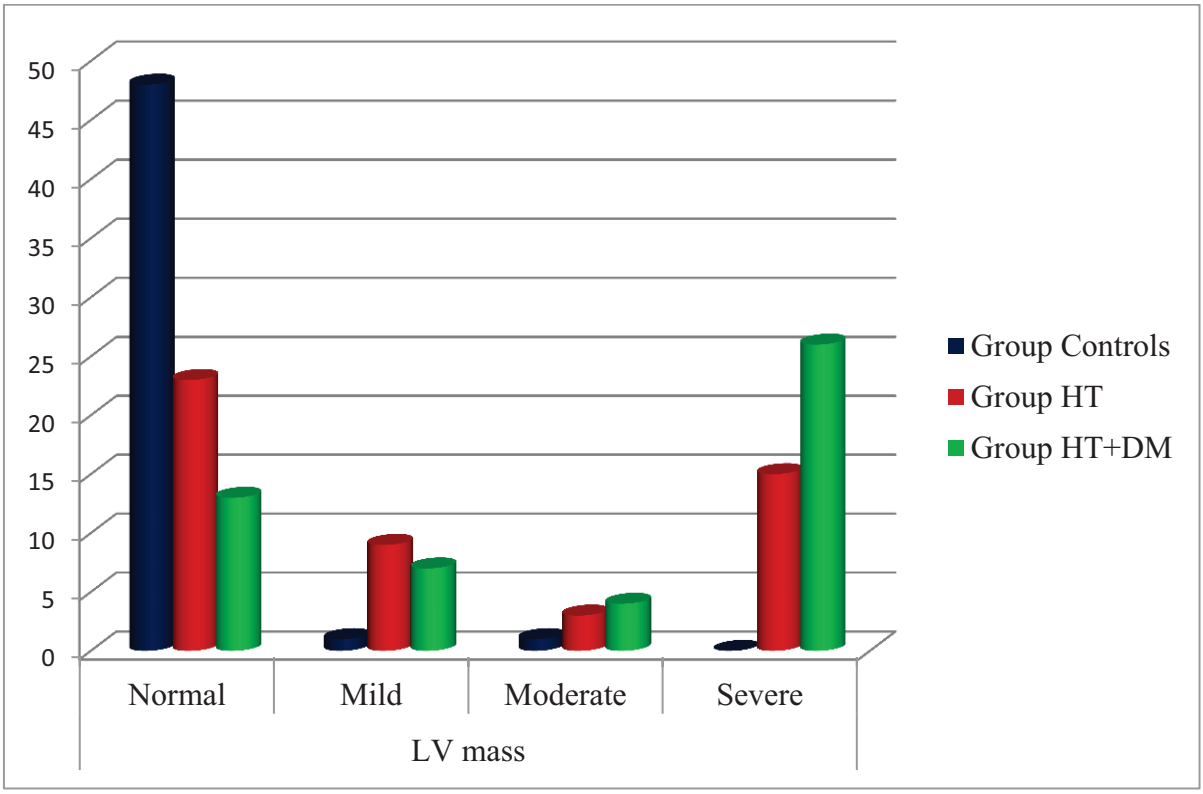


Table 19: Comparison of LV mass between two groups (HT, HT+DM):

			Group		Total
			HT	HT + DM	
LV mass	Normal	Count	23	13	36
		% within LV mass	63.9%	36.1%	100.0%
		% within Group	46.0%	26.0%	36.0%
	Mild	Count	9	7	16
		% within LV mass	56.3%	43.8%	100.0%
		% within Group	18.0%	14.0%	16.0%
	Moderate	Count	3	4	7
		% within LV mass	42.9%	57.1%	100.0%
		% within Group	6.0%	8.0%	7.0%
	Severe	Count	15	26	41
		% within LV mass	36.6%	63.4%	100.0%
		% within Group	30.0%	52.0%	41.0%
Total		Count	50	50	100
		% within LV mass	50.0%	50.0%	100.0%
		% within Group	100.0%	100.0%	100.0%

While comparing these two groups (HT, HT+DM) according to the severity of LV mass, the difference of LV mass was not statistically significant ($p = 0.106$). So we did subgroup analysis between male and female cases separately in these groups.

Table 20: Comparison LV mass between two groups (HT, HT+DM) in relation with Gender:

Sex				Group	
				HT	HT + DM
Male	LV mass	Normal	Count	12	8
			% within Group	52.2%	36.4%
		Mild	Count	4	4
			% within Group	17.4%	18.2%
		Moderate	Count	0	3
			% within Group	.0%	13.6%
		Severe	Count	7	7
			% within Group	30.4%	31.8%
Female	LV mass	Normal	Count	11	5
			% within Group	40.7%	17.9%
		Mild	Count	5	3
			% within Group	18.5%	10.7%
		Moderate	Count	3	1
			% within Group	11.1%	3.6%
		Severe	Count	8	19
			% within Group	29.6%	67.9%

The table 20&21 explains that there is significant statistical correlation (p=0.042) of LV mass between female hypertensive and both hypertensive diabetic group.

But there is no significant statistical correlation in males of HT and DM group (p = 0.286).

Table 21: Chi-Square test for Comparison LV mass between two groups (HT, HT+DM) in relation with Gender:

Sex		Value	Df	p value
Male	Pearson Chi-Square	3.780(a)	3	.286
	Likelihood Ratio	4.942	3	.176
	Linear-by-Linear Association	.673	1	.412
	N of Valid Cases	45		
Female	Pearson Chi-Square	8.216(b)	3	.042
	Likelihood Ratio	8.454	3	.038
	Linear-by-Linear Association	6.521	1	.011
	N of Valid Cases	55		

Table 22: Comparing the mean LV mass in between two groups (HT, HT+DM):

	Group	N	Mean	Std. Deviation	Std. Error Mean
LV mass	HT	50	188.7868	62.57260	8.84910
	HT + DM	50	214.0584	64.71982	9.15276

After comparing mean left ventricular mass between these two (HT, HT+DM) groups, this gives p value of 0.050 and not statistically significant. But mean LV mass is increasing in HT+DM group.

Table 23: Chi-Square Test for Comparison the mean LV mass in between two groups (HT, HT+DM):

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	T	Df	p value	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
LV mass	Equal variances assumed	.014	.908	-1.985	98	.050	-25.2716	12.73105	-50.53596	-.00724
	Equal variances not assumed			-1.985	97.889	.050	-25.2716	12.73105	-50.53632	-.00688

Table 24: Comparison of mean LV mass for males in between two groups (HT, HT+DM) (T-Test):

	Group	N	Mean	Std. Deviation	Std. Error Mean
LV mass	HT	23	211.2470	72.98173	15.21774
	H T+ DM	22	224.2382	70.07182	14.93936

After that subgroup analysis done between these two groups (HT, HT+DM) according to the gender which showed that the mean left ventricular mass for males in hypertension group was 211.25gm and in hypertension and diabetics group was 224.24gm with p value of 0.0546. This explains that mean

left ventricular mass was increasing in the second group (HT+DM) but there is no significant statistical correlation in males of these groups (HT, HT+DM) and LV mass.

Table 25: Chi-Square Test for Comparison of mean LV mass for males in between two groups (HT, HT+DM):

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	T	Df	p value	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
LV mass	Equal variances assumed	.155	.696	-.609	43	.546	-12.9912	21.34493	-56.03738	30.05493
	Equal variances not assumed			-.609	42.999	.546	-12.9912	21.32520	-55.99762	30.01517

Table 26: Comparison of mean LV mass for females in between two groups (HT, HT+DM) (T-Test):

	Group	N	Mean	Std. Deviation	Std. Error Mean
LV mass	HT	27	169.6541	45.22834	8.70420
	HT + DM	28	206.0600	60.26539	11.38909

Table 27: Chi-Square Test for Comparison of mean LV mass for females in between two groups (HT, HT+DM):

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	T	Df	p value	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
LV mass	Equal variances assumed	2.682	.107	-2.527	53	.015	-36.4059	14.40876	-65.30623	-7.50562
	Equal variances not assumed			-2.540	50.028	.014	-36.4059	14.33438	-65.19697	-7.61488

For females mean left ventricular mass for hypertension group was 169.65 and for hypertension and diabetic group was 206.06 with p value of 0.015. This explains that there is significant statistical correlation between females of these two groups (HT, HT+DM) and LV mass.

VII. DISCUSSION

In our study, we studied 150 subjects (50-HT only, 50- HT+DM, 50-Controlswithout HT and DM) and they were compared with LV mass. The minimum age of the cases and controls was 36 years and maximum age was 85 years. There is significant statistical correlation between age group and left ventricular mass in hypertension with diabetes group with the p value of 0.039. But in control group and cases with hypertension only group, the p value was > 0.05 and it was not statistically significant.

There is significant statistical correlation between female (p=0.042) cases with diabetes and hypertension and severity of abnormal left ventricular mass, than males (p=0.286).

There is no significant statistical correlation between body mass index, diastolic dysfunction, smoking and alcoholism with left ventricular mass.

There is no significant statistical correlation between duration of hypertension and diabetes with LV mass (P>0.05) But there is increase in mean LV mass in hypertension and diabetes group.

While comparing LV mass between these three groups (HT, HT+DM, Control) there is significant statistical correlation (p =0.001) between three groups. But while comparing the two groups (HT, HT+DM), the difference of LV mass according to the severity of abnormal LV mass was not statistically

significant. So we did subgroup analysis between male and female cases separately in these groups. There is significant statistical correlation (p=0.042) of LV mass between hypertensive and both hypertensive and diabetic group females. But there is no significant statistical correlation of LV mass in males in these two groups.

Then we took mean LV mass and compared between the two (HT, HT+DM) groups.

While comparing mean left ventricular mass between these two (HT, HT+DM) groups, there is no significant statistical correlation (p=0.05) between LV mass and these groups.

After that subgroup analysis done between these two groups (HT, HT+DM) according to the gender which showed that the mean left ventricular mass for males in hypertension group was 211.25gm and in hypertension and diabetics group it was 224.24gm. This explains that mean left ventricular mass was increasing in the second group (HT+DM) but not correlated statistically (p=0.546).

For females mean left ventricular mass for hypertension group was 169.65 and for hypertension and diabetic group was 206.06. This explains that there is significant statistical (p=0.015) correlation of increased LV mass in diabetes and hypertension group.

In The Framingham study [20], they studied 2623 diabetic subjects out of which 1514 were women with mean age of 53 years without cardiac disease and they found that women more left ventricular mass compared to men with p value of < 0.001 for women and p=0.054 for men. They explained that the increased activation of serine/threonine protein kinase which is an inhibitor of apoptosis and also the oestrogen receptors in cardiomyocytes in females are the responsibility of increased LV mass in females.

Maurizio Galderisi [78] and colleagues studied 2529 women and 1986 men with diabetes and without cardiac disease, which shows that increased left ventricular mass in diabetic women with a p value of <0.05.

Alexander Tenenbaum and their colleagues [76] studied 550 hypertensive (314 men, 246 women), 200 of them (108 men, 92 women) was type 2 diabetes mellitus. They found that female with diabetes and hypertension has higher prevalence of increased left ventricular mass compared to males.

The Augsburg family study [77] showed that increased left ventricular mass in female diabetics.

VIII. CONCLUSION

The study shows that there is increased left ventricular mass in females with hypertension and diabetes mellitus when compared to females with hypertension alone.

There is no significant difference in the left ventricular mass in males in between the two (HT, HT+DM) groups.

But the mean left ventricular mass was increased in both males and females of diabetes and hypertension group when compared to hypertension only group.

The increased duration of hypertension and diabetes is associated with increased mean left ventricular mass in cases of both hypertension and diabetes group.

In our study, advancing age group is associated with increased left ventricular mass in cases of both hypertension and diabetes group.

Hence, increased left ventricular mass is the important cause for increased cardiovascular morbidity and mortality, the females with diabetes and hypertension should be managed aggressively for reduction of left ventricular mass.

LIMITATIONS OF THE STUDY

1. Study population is small.
2. As echocardiography has observer variation, the same results need not be obtained in other similar studies.
3. The duration of undiagnosed diabetes mellitus and hypertension in the patient who presented towards and taken into the study is not taken into consideration.

IX. ANNEXURE

IX. a. BIBLIOGRAPHY:

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IX. b. PROFORMA:

NAME:

AGE:

SEX:

ADDRESS: OP No:

HISTORY AND PAST HISTORY:

H/O Hypertension:

H/O Diabetes Mellitus:

H/O Coronary Artery Disease:

H/O Valvular Heart Disease:

H/O Chronic Kidney Disease:

Drug History:

GENERAL EXAMINATION:

Built: Pallor: Icterus:

Pedal oedema: Temp: Hydration:

Clubbing: PR: BP: BMI:

Cardiovascular System:

Respiratory System:

Abdomen:

Central Nervous System:

Investigations:

Blood:

Complete Hemogram:

Hb: TC: DC: P: L: M: E: ESR:

FBS:

PPBS:

B.Urea:

S.Creatinine:

Serum electrolytes: Na: K+:

Total Cholesterol:

Triglyceride level:

Urine:

Sugar:

Albumin:

Deposits:

ECG:

Echocardiography:

LVIDD:

PWTD:

IVSTD:

EF:

Valves:

Others:

LV mass (penn) =1.04[(LVIDD+IVST-D+PWT-D)^3 – (LVIDD)^3] – 13.6gm.

Chest X-Ray PA view

IX. c. ABBREVATIONS:

HT - Hypertension

DM - Diabetes mellitus

LV mass – Left ventricular mass

LVIDD - Left ventricular internal diameter in diastole

IVST- D – Interventricular septal thickness in diastole

PWT- D – Posterior wall thickness in diastole

2-D Echo – Two dimensional echocardiography

ECG - Electrocardiography

AT1- Angiotensin receptor 1

AT II – Angiotensin receptor II

RAAS – Renin-Angiotensin Aldosterone System

PA view – Postero Anterior view

RWT- Regional Wall Thickness

EF – Ejection Fraction

PR – Pulse Rate

BP – Blood Pressure

BMI – Body Mass Index

DD – Diastolic Dysfunction

FBS – Fasting Blood Sugar

PPBS – Post Prandial Blood Sugar

PPAR – Peroxisome Proliferated Activated Receptor

ERK – Extracellular singal Regulated Kinase pathways

PKC – Phosphokinase C

VEGF – Vascular Endothelial Growth Factor

IGF – Insulin-like Growth Factor

MHC – Myosin Heavy Chain

NO – Nitric Oxide

ROS – Reactive Oxygen Species

AGEs – Advanced Glycated End products

IGT – Impaired Glucose Tolerance

MI – Myocardial Infarction

NE – Nor Epinephrine

ACE – Angiotensin Converting Enzyme

JNC – Joint National Committee

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.1223/ME-1/Ethics/2013 Dt:07.03.2013.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on comparison of left ventricular mass in hypertensive patients with diabetes mellitus and without diabetes mellitus" for project work submitted by Dr. S.Krishnamoorthi,MD, General Medicine PG Student, Kilpauk Medical College, Chennai.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

19/4/13.

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

GOVT. KILPAUK MEDICAL COLLEGE
19 APR 2013
Kilpauk, Chennai-10.

MASTER CHART FOR HYPERTENSION WITHOUT DIABETES MEELITUS GROUP:

Sl.No	Name	HT op no	Age	Sex	Religion	BMI	HT duration	Smoking	Alcoholism	BP	LVID	IVST	PWT	LV mass	EF	DD
1	Chinnaiah	1325887	72	Male	Hindu	23.88	6	Yes	No	140/90	3.95	1.53	1.06	213.22	60	1
2	Gopalakrishnan	2334/11	42	Male	Hindu	24.61	6	No	No	130/90	4.38	1.44	1.2	258.8	64	No
3	Ramadoss	1184/12	73	Male	Hindu	23.18	7	No	No	140/90	5	1.4	1.1	295.15	74	1
4	Rukmani	913/05	57	Female	Hindu	24.34	11	No	No	130/80	4.67	1.1	0.8	175.42	65	No
5	Muthaiah	1326/13	70	Male	Hindu	23.23	6	Yes	Yes	140/90	4.23	1.12	0.9	161.59	60	No
6	kaliammal	971/09	60	Female	Hindu	23.53	6	No	No	130/80	3.9	1.73	1.3	270.83	60	1
7	Indrani	689/08	60	Female	Hindu	24.84	17	No	No	110/80	3.6	1.1	0.8	110.91	74	No
8	Balaraman	32/10	68	Male	Hindu	24.98	7	No	No	130/80	4.9	1.53	1.37	357.58	64	No
9	Rajamani	2245/06	50	Male	Hindu	27.34	7	No	No	140/80	4.2	1.1	1	169.4	65	No
10	Perumal	1315/10	70	Male	Hindu	23.14	8	No	No	140/90	4.9	1.7	1	320.58	65	1
11	Jayaraman	2030/10	60	Male	Hindu	25.91	12	Yes	Yes	140/90	3.87	1.66	1.32	260.4	72	No
12	koneri	654/05	85	Male	Hindu	27.34	12	No	No	140/90	3.65	2.08	1.44	319.17	78	1
13	Pandian	331/08	58	Male	Hindu	23.88	20	No	Yes	130/90	3.02	1.4	0.93	117.01	70	1
14	Umarani	216/08	50	Female	Christian	23.05	8	No	No	140/90	4.7	1.35	1.01	244.4	76	1
15	Moorthy	234/05	42	Male	Hindu	22.49	8	No	No	140/80	4.3	0.8	0.8	117.31	65	1
16	Doss	6853/07	70	Male	Christian	23.53	7	No	No	140/90	4	1.5	0.7	167.7	80	1
17	Raman	507/08	75	Male	Hindu	25.71	6	No	No	140/80	5.24	1.13	0.73	208.99	60	1
18	Usha	2305/06	54	Female	Hindu	24.97	7	No	No	130/80	3.5	1.18	0.7	103.76	65	1
19	Dinesh	2431/08	43	Male	Hindu	25.39	6	Yes	Yes	140/90	4.76	1.04	0.64	152.01	69	No
20	Lakshmi	316/08	70	Female	Hindu	28	6	No	No	130/80	4.9	1.31	0.73	211.67	60	1
21	Manonmani	717/11	65	Female	Hindu	26.84	6	No	No	130/90	4.3	1	0.9	151.57	78	1
22	Selvi	421/03	41	Female	Hindu	25.78	11	No	No	130/90	3.1	1.02	1.1	103.34	66	No
23	Sivagami	1784/09	45	Female	Hindu	22.83	6	No	No	140/90	3.97	1.1	0.9	142.61	72	1
24	Amir	574/03	60	Male	Muslim	28.13	11	Yes	Yes	140/90	5.2	1.1	0.8	212.4	72	1
25	Somasundaram	838/08	42	Male	Hindu	24.98	6	No	No	130/90	4.53	2.2	0.8	333.76	65	1

26	Krishnamoorthi	1954/13	52	Male	Hindu	24.22	7	Yes	Yes	140/90	4.6	1.1	0.6	145.22	70	No
27	Harikrishnan	195/12	55	Male	Hindu	24.68	6	No	Yes	140/90	4.96	0.95	0.7	159.85	60	1
28	Gowri	99/07	55	Female	Hindu	11	11	No	Yes	140/90	4.68	0.95	0.75	149.88	60	1
29	Abdhul Wahib	1700/10	67	Male	Muslim	24.98	6	Yes	No	130/90	4.62	1.16	0.92	196.64	70	1
30	Lakshmi	251/12	45	Female	Hindu	24.97	6	No	No	130/80	4.3	1.25	0.96	190.64	60	1
31	Vasu	412/12	50	Male	Hindu	24.91	6	Yes	No	140/80	4.96	0.87	0.58	133.4	65	1
32	Babu	501/09	63	Male	Hindu	25.71	6	Yes	Yes	140/70	5.15	0.87	0.63	150.19	67	1
33	Pamana	150/10	47	Female	Hindu	28.89	6	No	No	120/80	5.04	1.06	0.58	163.26	67	1
34	Chandra	1493/07	50	Female	Hindu	28.67	6	No	No	`130/80	4.58	1.06	0.67	147.77	67	1
35	Shajahan	193/09	50	Male	Muslim	25.71	6	No	No	130/80	4.78	1.3	0.9	226.49	69	1
36	Fazudein	324/05	73	Male	Muslim	25.91	16	No	No	140/90	5.2	1	0.7	181.82	73	1
37	Malliga	1136/13	50	Female	Hindu	27.53	20	No	No	130/80	4.3	0.96	0.86	142.1	65	No
38	Mehroon	2177/05	52	Female	Muslim	26.04	8	No	No	120/80	4.2	1	0.8	133.99	63	1
39	Vennila	261/09	70	Female	Hindu	27.64	8	No	No	130/80	3.8	1.1	0.9	132.25	60	No
40	Rajamani	477/05	74	Female	Hindu	27.34	8	No	No	120/80	4.7	1.1	0.7	164.03	65	No
41	Rosy	496/10	60	Female	Christian	27.24	6	No	No	120/80	4.5	1	0.8	151.68	65	1
42	Muniammal	1529/13	50	Female	Hindu	25.48	6	No	No	130/80	4.8	1.3	0.9	228.1	68	No
43	Thangamani	296/01	75	Female	Hindu	25.39	8	No	No	130/80	5.1	1.1	0.8	205.16	70	1
44	Sakunthala	193/12	59	Female	Hindu	24.01	6	No	No	140/90	3.9	1.2	0.9	149.35	65	1
45	Mysiammal	480/06	50	Female	Hindu	24.03	7	No	No	140/80	4.5	1.1	0.9	177.24	67	No
46	Ansar Bee	1856/06	50	Female	Muslim	25.56	8	No	No	130/70	4.7	1.3	0.9	220.07	70	No
47	Valsala	1166/07	39	Female	Hindu	23.53	6	No	No	110/80	4	1	0.8	122.76	65	No
48	Regina	2947/07	47	Female	Christian	22.55	6	No	No	120/80	3.9	1.3	0.9	160.77	65	No
49	Lakshmi	366/12	65	Female	Hindu	26.67	6	No	No	140/70	4.4	1.7	0.9	254.53	70	No
50	Kanagavalli	2027/13	73	Female	Hindu	23.53	6	No	No	130/80	3.9	1.4	0.9	172.57	68	No

MASTER CHART FOR HYPERTENSION WITH DIABETES MELLITUS GROUP:

Sl.No	Name	HT.OP.No	Age	Sex	Religion	BMI	Duration of HT	Duration of DM	Smoking	Alcoholism	BP	FBS	PPBS	LVID	IVST	PWT	LV mass	EF	DD
1	Kannan	1366/02	70	Male	Hindu	24.98	6	6	No	No	130/90	110	150	5.1	1.5	1.3	361.2	60	No
2	Radhakrishnan	1325942	58	Male	Hindu	24.8	16	16	Yes	No	130/80	120	165	5.28	1.36	1.18	330.65	63	1
3	Mariammal	1325635	60	Female	Hindu	26.04	16	16	No	No	140/90	130	190	4.79	1.07	0.6	152.47	60	1
4	Kanniyammal	1326847	65	Female	Hindu	28.25	7	8	No	No	140/90	128	198	3.9	1.63	1.38	267.85	65	1
5	Meena	1326969	40	Female	Hindu	23.83	9	8	No	No	130/90	108	156	5.2	1.75	0.8	324.27	60	1
6	Samuvel	719/02	81	Male	Christian	25.21	6	7	No	No	130/90	120	200	5.02	1.78	1.16	379.37	70	No
7	Nagapushpam	647/10	57	Female	Hindu	24.13	8	6	No	No	130/84	114	164	4.39	0.87	0.77	126.44	60	1
8	Maheswari	1019/02	50	Female	Hindu	24.24	11	13	No	No	140/78	126	178	5.25	1.16	0.82	228.96	60	1
9	Palani	13152123	61	Male	Hindu	24.16	12	11	No	No	140/84	132	184	5.2	1.12	0.92	234.85	60	1
10	Thangarajan	1232/11	72	Male	Hindu	25.71	9	6	Yes	Yes	130/86	110	174	4.3	1.11	1.01	178.91	60	1
11	Vasudevan	703/10	52	Male	Hindu	25.28	15	13	Yes	Yes	136/94	118	176	3.1	1.59	1.78	237.09	89	1
12	Saraswathi	2393/13	60	Female	Hindu	25.89	11	11	No	No	130/80	128	186	3.3	1.73	1.06	183.93	64	1
13	Sankar	208/03	45	Male	Hindu	25.65	10	6	Yes	Yes	140/90	130	194	4.76	0.98	0.94	184.24	65	1
14	Amsa	767/10	60	Female	Hindu	28.67	15	12	No	No	130/84	114	202	4.33	1.15	0.6	135.72	60	1
15	Rajam	1171/08	56	Female	Hindu	26.37	6	6	No	No	140/78	108	208	4.97	1.57	0.64	243.68	74	1
16	Subathra	1559/06	51	Female	Christian	26.22	13	13	No	No	134/70	76	146	5.2	1.5	0.9	296.7	60	1
17	Ranjitham	1365/08	50	Female	Hindu	23.44	6	6	No	No	128/92	114	172	4.7	1.1	0.7	164.03	67	1
18	Nirmala	972/04	45	Female	Christian	24.03	6	6	No	No	130/92	120	160	4.49	1.14	0.92	184.51	67	1
19	mani	3827/06	45	Male	Hindu	24.38	7	6	No	Yes	128/88	130	170	3.97	1.02	1.13	159.72	71	No
20	Subramaniyan	424/12	73	Male	Hindu	24.16	6	10	No	No	130/90	126	172	4.9	1.11	0.9	207.18	72	No
21	Muthukumar	2481/13	58	Male	Hindu	25.82	6	6	No	No	140/90	130	200	4.14	1.3	1.1	203.52	67	No
22	Varadhamma	937/10	65	Female	Hindu	27.99	7	7	No	No	130/80	110	160	4.9	1.3	0.9	236.27	65	No
23	Bangarammal	1484/13	66	Female	Hindu	28.76	7	10	No	No	120/80	106	156	4.8	1.1	1	213.03	67	1
24	Kuyilammal	844/08	60	Female	Hindu	27.27	6	6	No	No	130/80	110	170	3.9	1.3	1.1	184.76	65	1

25	Vanaja	2373/08	55	Female	Hindu	22.76	7	7	No	No	120/80	120	180	4.1	1.5	1	213.72	63	1
26	Vijaya	910/08	62	Female	Hindu	25.72	11	15	No	No	140/90	118	168	4	0.8	0.8	102.48	65	1
27	K.R.Ravi	817/12	51	Male	Hindu	25.82	10	10	No	Yes	130/90	130	190	4.5	0.8	0.7	116.27	62	1
28	Ayyappan	274/07	64	Male	Hindu	24.98	6	10	Yes	No	120/80	120	170	5	1.2	1.1	260.98	60	1
29	Vijaya	907/10	43	Female	Hindu	26.56	7	7	No	No	130/90	110	170	3.9	0.9	0.6	88.47	68	No
30	Mohan	1519/07	54	Male	Hindu	23.81	6	6	No	Yes	140/90	120	170	4.6	1.1	0.9	184.17	69	No
31	Venkatesan	2447/09	50	Male	Hindu	24.22	20	16	No	No	120/80	117	178	4.8	0.9	0.9	170.38	70	1
32	Kasi	2520/08	60	Male	Hindu	26.85	6	7	No	No	140/80	120	170	4.9	1.25	1.17	271.96	65	No
33	Emely	2738/08	60	Female	Christian	25.56	6	10	No	No	120/80	118	188	5	1.2	1	244.58	67	1
34	Vengammal	1582/07	65	Female	Hindu	23.53	6	6	No	No	130/80	130	170	5.1	1.1	0.9	220.67	62	1
35	Parvathy	383/09	55	Female	Hindu	23.73	6	6	No	No	130/80	108	178	4.6	1.04	0.9	176.09	67	No
36	Krishnan	817/06	52	Male	Hindu	23.88	6	6	No	Yes	140/90	130	190	4.6	0.95	0.86	159.08	62	1
37	mani	1342/03	65	Male	Hindu	25.1	20	16	No	No	120/80	120	170	4.9	1.2	0.78	202.73	67	1
38	kanniyammal	822/08	50	Female	Hindu	24.84	6	6	No	No	120/80	130	170	4.2	1.15	0.82	153.63	63	1
39	Thangam	4244/07	64	Female	Hindu	23.05	8	10	No	No	120/80	125	175	5.2	1.3	0.9	261.6	64	No
40	Shantha	1510/06	57	Female	Hindu	25.48	6	6	No	No	140/80	126	190	4.3	1.4	0.9	202.71	60	1
41	Pattu	652/10	70	Female	Hindu	23.73	6	6	No	No	130/90	108	178	5.2	1.32	1	282.44	65	No
42	Suleka banu	1945/07	45	Female	Hindu	22.76	6	6	No	No	140/90	110	170	4.2	1	0.8	133.99	67	No
43	Parimal Seivi	5315/06	53	Female	Hindu	24.44	7	7	No	No	130/80	120	190	4.7	1.23	1	224.55	63	1
44	Angappan	1377/08	80	Male	Hindu	25.56	6	6	No	No	120/80	116	156	4.9	1.23	1.15	265.31	60	No
45	Veerabathran	1313/10	45	Male	Hindu	24.01	11	11	Yes	No	140/90	128	190	3.9	1	0.8	117.31	67	1
46	Pushparani	572/07	58	Female	Hindu	27.41	12	15	No	No	130/80	100	150	4.7	1.3	1	235.14	63	1
47	Sethuraman	2044/13	56	Male	Hindu	25.51	13	15	No	No	120/80	96	160	5.1	1.3	1	269.88	63	1
48	Lakshmipathy	2450/03	65	Male	Hindu	25.46	11	12	No	No	130/90	126	180	5.1	1	0.9	205.16	62	No
49	Nagammal	211/06	75	Female	Hindu	27.94	16	16	No	No	130/90	116	140	5.23	1.23	1.1	286.99	60	1
50	Thanikachalam	1322/68	68	Male	Hindu	23.81	16	16	No	No	120/80	108	132	4.5	1.5	0.9	233.28	63	No

MASTER CHART FOR CONTROLS GROUP:

Sl.No	Name	Med. OP No	Age	Sex	Religion	BMI	Duration of HT	Duration of DM	Smoking	Alcoholism	BP	FBS	PPBS	LVID	IVST	PWT	LV mass	EF	DD
1	Lakshmanan	231	43	Male	Hindu	22.1	No	No	Yes	No	120/80	100	136	4.6	0.7	0.6	98.76	60	No
2	Saradha bai	232	57	Female	Hindu	27.77	No	No	No	No	130/80	90	121	4.1	0.7	0.65	83.08	66	No
3	Thilagavathi	201	51	Female	Hindu	26.84	No	No	No	No	130/80	108	123	3.8	0.7	0.72	77.26	70	No
4	Sundari devi	220	37	Female	Hindu	25.08	No	No	No	No	110/80	110	132	3.9	0.75	0.65	79.54	76	No
5	Sampath	152	50	Male	Hindu	26.84	No	No	Yes	Yes	120/80	104	134	4.6	0.81	0.61	112.06	64	No
6	Jothi	190	50	Female	Hindu	21.47	No	No	No	No	100/70	86	112	3.7	0.78	0.71	79.11	67	No
7	vengaiah	169	63	Male	Hindu	23.29	No	No	No	No	120/80	98	122	3.6	0.72	0.61	62.49	72	No
8	Thanigaimalai	199	49	Male	Hindu	28.74	No	No	Yes	No	130/80	78	123	5	0.7	0.6	116.45	68	1
9	Usha	218	53	Female	Hindu	24.69	No	No	No	No	120/80	90	134	4.5	0.6	0.56	80.2	69	No
10	Janaki	209	52	Female	Hindu	28.23	No	No	No	No	110/70	86	138	5.2	1	0.7	181.82	70	1
11	Kothai	202	37	Female	Hindu	28.54	No	No	No	No	120/80	120	142	4.2	0.7	0.6	82.38	65	No
12	David selvaraj	208	44	Male	Christian	24.31	No	No	Yes	No	120/80	116	133	4.4	0.8	0.71	112.49	64	No
13	Devika	240	38	Female	Hindu	25.39	No	No	No	No	110/70	100	120	4.2	0.71	0.65	88.1	68	No
14	Surgunavathi	187	46	Female	Hindu	24.22	No	No	No	No	120/80	78	134	4.2	0.7	0.6	82.38	72	No
15	Ashok kumar	223	56	Male	Hindu	24.61	No	No	No	No	110/80	84	126	5.1	1.1	0.9	220.67	74	1
16	Radha	170	37	Female	Hindu	24.46	No	No	No	No	110/70	98	128	4.1	0.8	0.6	87.75	68	No
17	Kalavathi	141	49	Female	Hindu	24.24	No	No	No	No	120/80	90	108	3.9	0.8	0.7	88.47	69	No
18	Jayanthi	140	49	Female	Hindu	24.03	No	No	No	No	120/70	84	110	3.8	0.72	0.62	70.56	70	No
19	Baskaran	139	53	Male	Hindu	23.8	No	No	No	No	130/80	78	112	4.2	0.9	0.6	101.95	72	No
20	Ranganathan	147	54	Male	Hindu	24.61	No	No	No	No	130/80	90	124	4.6	0.8	0.76	128.27	76	No
21	Nandhini	146	47	Female	Hindu	23.83	No	No	No	No	120/80	124	140	4.1	0.8	0.75	102.3	74	No

22	Esther	149	48	Female	Christian	23.63	No	No	No	No	130/80	108	128	4.2	0.8	0.68	99.93	75	No
23	Mahesh	157	41	Male	Hindu	23.53	No	No	No	No	110/80	120	138	4.3	0.9	0.6	106.63	79	No
24	Lakshmaiah	164	50	Male	Hindu	25	No	No	No	Yes	130/86	112	136	4.7	0.8	0.6	114.48	78	No
25	Ramamoorthy	165	48	Male	Hindu	23.62	No	No	No	Yes	110/78	114	132	4.5	0.8	0.8	127.69	77	No
26	Gopal	161	49	Male	Hindu	25.64	No	No	No	No	128/88	118	134	4.9	0.85	0.75	149.66	65	No
27	Muthukumar	168	50	Male	Hindu	24.61	No	No	No	No	120/80	108	124	4.7	0.82	0.72	131.11	67	No
28	Raj	171	50	Male	Hindu	26.56	No	No	No	Yes	130/80	92	114	4.1	0.8	0.66	93.48	67	No
29	Janaki	162	57	Female	Hindu	24.24	No	No	No	No	130/80	86	112	3.6	0.76	0.7	84.86	68	No
30	Meena	160	37	Female	Hindu	22.6	No	No	No	No	110/80	96	128	4.4	0.8	0.78	120.21	66	No
31	Seetharam	192	58	Male	Hindu	25.39	No	No	No	No	120/82	104	138	4.9	0.9	0.6	136.67	65	No
32	Rajan	193	59	Male	Hindu	27.18	No	No	No	Yes	130/88	121	140	4.3	0.76	0.65	97.33	69	No
33	Kasthuri	194	55	Female	Hindu	23.63	No	No	No	No	128/84	112	130	4.1	0.7	0.65	83.08	66	No
34	Chandra	196	45	Female	Hindu	24.65	No	No	No	No	130/80	117	134	4.2	0.72	0.68	91.99	65	No
35	Vasanthi	174	50	Female	Hindu	26.22	No	No	No	No	136/86	109	138	4	0.76	0.7	89.12	65	No
36	Anbu	233	53	Female	Hindu	23.44	No	No	No	No	130/80	100	128	4.5	0.91	0.65	123.08	65	No
37	Suseela	228	36	Female	Hindu	22.6	No	No	No	No	110/80	100	138	4	0.8	0.7	92.87	65	No
38	Hemalatha	226	50	Female	Hindu	24.34	No	No	No	No	120/80	102	128	4.2	0.95	0.75	122.94	65	1
39	Jayalakshmi	221	50	Female	Hindu	25.39	No	No	No	No	110/80	86	126	4.1	0.7	0.7	87.75	67	No
40	Manohari	216	51	Female	Hindu	24.46	No	No	Yes	No	124/84	94	128	3.9	0.8	0.7	88.47	68	No
41	Sundar	207	47	Male	Hindu	25.3	No	No	No	No	114/78	120	134	4.5	0.79	0.7	115.15	68	No
42	Vijay Anand	213	41	Male	Christian	24.38	No	No	No	No	112/76	114	136	4.7	0.8	0.7	126.29	68	No
43	Venkatesan	211	41	Male	Hindu	24.45	No	No	No	Yes	118/80	102	128	4.8	0.85	0.75	144.01	65	No
44	Chinthamani	112	49	Female	Hindu	23.71	No	No	No	No	114/78	108	130	4.4	0.86	0.77	125.84	65	No
45	Kusala	115	52	Female	Hindu	23.42	No	No	No	No	120/78	110	120	3.9	0.78	0.7	86.66	69	1
46	Vasantha	116	56	Female	Hindu	24.88	No	No	No	No	134/84	90	130	4	0.8	0.6	83.6	66	No
47	Tamilselvan	119	55	Male	Hindu	25.07	No	No	No	No	120/70	92	136	3.7	0.87	0.74	89.43	65	No
48	Suresh	120	38	Male	Hindu	26.29	No	No	Yes	No	120/80	86	128	4.1	0.8	0.7	97.36	67	No
49	Padmini	143	59	Female	Hindu	24.69	No	No	No	h	110/70	102	128	4	0.8	0.75	97.63	65	No
50	Prabavathy	234	60	Female	Hindu	26.91	No	No	No	No	110/76	92	138	4.1	0.82	0.72	101.3	70	1

Abbreviations Used in MASTER CHART:

HT	- Hypertension
DM	- Diabetes Mellitus
BMI	- Body Mass Index
LV mass	- Left Ventricular Mass
IVST	- InterVentricular Septal Thickness
PWT	- Posterior Wall Thickness
LVID	- Left Ventricular Internal Diameter
EF	- Ejection Fraction
DD	- Diastolic Dysfunction
FBS	- Fasting Blood Sugar
PPBS	- Post Prandial Blood Sugar
FBS	- Fasting Blood Sugar
BP	- Blood Pressure